Comparison of the Dietary Intake of the Urban Black Population in Cape Town in 1990 with 2009

by
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DECLARATION

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ABSTRACT

Background: In 1990 the BRISK was undertaken in the black population in five townships in Cape Town, Langa, Gugulethu, Khayalitsha, Nyanga, and Crossroads. The prevalence of non-communicable diseases (diabetes and heart disease) and their risk factors, including dietary intake were investigated.

Aim: To determine the dietary intake of the urban black population (25–64 years) of Cape Town in 2009 and compare these findings with a similar sample examined in the same townships in 1990.

Study design and sample: A cross-sectional survey including a representative sample of 392 males and 707 females (n=1099) was drawn from the same townships as in 1990.

Methods: Socio-demographic data were collected by trained field workers. Weight, height, and waist and hip circumference were measured. Body mass index (BMI), and waist- hip ratio (WHR) were calculated. Dietary intake data (macro- and micronutrients, mean adequacy ratio (MAR), food groups and portion sizes) were calculated using the MRC FoodFinder program and compared with the dietary reference intakes. The MAR was calculated for each participant.

Data analyses: Anthropometric and dietary data are presented as means and standard deviations by age, level education, type of housing, degree of urbanisation and asset index (proxy for socio-economic status). Associations of dietary data with anthropometric and biochemistry data (TC, HDL-C, LDL-C, and glucose), blood pressure, asset index, and degree of urbanisation were assessed. A linear regression model was computed using MAR as the dependent variable and adding age, gender, urbanisation, asset index and other variables to the model. Correlations between the asset index and urbanisation duration were done with energy and nutrient intakes using Pearson's correlations. Regressions were done to test the significance of various variables.

Results: Most of the adults had an education of at least 8–12 years, though 60% of the males and 58.3% of females were unemployed, and 13.8% were pensioners. Twenty-one per cent lived in formal houses, 35.4% in council houses or hostels, and 43.6% in shacks. Only 12.1% of adults had spent less than 20% of their life in an urban area. Thirty three per cent were classified as falling within the poorest tertile of the asset index. The percentage of adults with a BMI greater than 30 kg/m^2 was 63.3% in females and 12% in males. Males had a WC and WHR of 85.7 cm and 0.89 and females 97.5 cm and 0.85, respectively.

Analysis of the 24-hour dietary recall data showed very low mean energy intakes [M 6516 (2929); F 5760 (2446) kJ]. These results did not support the high prevalence of obesity, particularly in females, and also in four other studies among urban black populations. Hence the Goldberg equation was used to remove under-reporters. The remaining sample comprised 544 (214 males and 330 females) participants residing in Khayelitsha (42.4%), Langa (31.4%), Gugulethu (15.3%), and less than 10% in Crossroads and Nyanga.

After removal of the under-reporters, mean energy intakes were similar to those of the 1990 study and in 25–44-year-old males were 8557 kJ compared to 8500 kJ in 1990. In females they were 7619 kJ compared with 6400 kJ in 1990. Fat intakes were highest in 25–44-year-old males (32% energy [E]) and females (33.4% E) in 2009 compared to 1990 (males: 25.9% E, females: 27.0% E). Carbohydrate intakes were lower in 2009 (males 53.2% E, females: 55.5% E) than in 1990 (males: 61.3% E; females: 62% E), while sugar intake increased significantly (p <0.01) in females. There were significant positive correlations between urbanisation and total fat (p=0.016), saturated fat (p=0.001), monounsaturated fat (p=0.002) and fat as a %E intake (p=0.046). Urbanisation was inversely associated with intake of carbohydrate %E (p <0.001). Overall micronutrient intakes improved significantly compared to 1990 with the exception of calcium intake. This is thought to be due to the national fortification of maize and wheat flour. Of note is that energy and macronutrient intakes were all significant in a linear regression model using the MAR, as was duration of urbanisation.

The higher fat and lower carbohydrate %E intakes in this population demonstrate a transition to a more urbanised diet over the two decades. Forward regression was done to identify the significant variables which were then entered into a linear regression model. In males, BMI was significantly associated with protein intake, while in females BMI was related to saturated fat, saturated fat %E, carbohydrate, and carbohydrate %E. In terms of the duration of urbanisation it can be noted that carbohydrate %E, total fat %E energy, calcium, animal protein and total cholesterol were significant in males. In females, protein %E, carbohydrate %E, fat %E were significantly associated with greater urbanisation.

In the 2009 study, milk products, the meat group, legumes, SF and brick margarine in males and females were found to be statistically lower than those in the 1990 study. This was also found in the cereal group, but only for the males. Despite this, the 2009 study for the males and females, showed significantly higher intakes of eggs, vitamin C rich fruits and vegetables and PUFA sources compared to the 1990 group. This was also found in the cereal group for females. The percentage consumers for red meat, white meat, eggs, vegetables and fruit, and cereals increased from 1990 to 2009. For the dairy and fat groups, the percentage consumers decreased.

Conclusion: The nutrient intakes demonstrate that while certain changes have taken place between 1990 and 2009, the dietary pattern regarding foods eaten remains poor. The diet has become more urbanised and atherogenic with regard to fat distribution and carbohydrate intake, while the consumption of certain food groups has remained low, such as the poor consumption of dairy products and low intake of fruit and vegetables. However, overall mean micronutrient intakes increased to above the dietary reference intakes with the exception of calcium intake.

Recommendation: Interventions are urgently needed to combat the shift towards a continuing atherogenic diet and to improving the consumption of priority food groups, such as dairy products, and fruit and vegetables.

OPSOMMING

Agtergrond: In 1990 is die BRISK-studie onder die swart bevolking in vyf Kaapstadse woongebiede, Langa, Gugulethu, Khayalitsha, Nyanga, en Crossroads onderneem. Die voorkoms van nie-oordraagbare siektes (diabetes en hartsiekte) en hulle risikofaktore, insluitend dieetinname is ondersoek.

Doel: Om die dieetinname van die stedelike swart bevolking (25–64 jaar) van Kaapstad in 2009 te bepaal en hierdie bevindings met ’n soortgelyke steekproef uit dieselfde woongebiede as in 1990 te vergelyk.

Studieontwerp en steekproef: ’n Deursnee-opname wat ’n verteenwoordigende steekproef van 392 mans en 707 vroue (n=1099) insluit, is getrek vanuit dieselfde woongebiede as in 1990.

Metode: Sosiodemografiese data is deur opgeleide veldwerkers ingesamel. Gewig, lengte, en middel- en heupomtrek is gemeet. Die liggaamsmassa indeks (LMI) en middel- en heupverhouding (MHV) is bereken. Dieetinname data (makro- en mikronutriënte, gemiddelde toereikendheidsverhouding, voedselgroepe en porsiegroottes) is bereken met behulp van die *MRC FoodFinder*-program en vergelyk met die dieetverwysingsinnames. Die gemiddelde toereikendheidsverhouding is vir elke deelnemer bereken.

Data analises: Antropometrie en dieetdata word as gemiddeldes en standaardafwykings volgens ouderdom, onderwysvlak, soort behuising, mate van verstedeliking en bate indeks (volmag vir ekonomiese status) aangebied. Assosiasies van dieetdata met antropometrie en biochemiese data (TC, HDL-C, LDL-C, en glukose), bloeddruk, die bate indeks en mate van verstedeliking is getakseer. ’n Lineêre regressiemodel is bereken met behulp van die gemiddelde toereikendheidsverhouding as afhanklike veranderlike en byvoeging van ouderdom, geslag, verstedeliking, bate indeks en ander veranderlikes tot die model. Korrelasies tussen bate indeks en duur van verstedeliking met energie en nutriëntinnames is met behulp van Pearson se korrelasies gedoen. Regressies is gedoen om die betekenisvolheid van verskeie veranderlikes te toets.

Resultate: Die meeste volwassenes het ten minste 8–12 jaar opvoeding gehad alhoewel 60% mans en 58.3% vroue werkloos was, met 13.8% pensionarisse. Een-en-twintig persent het in formele huise gewoon, 35.4% in munisipale huise of hostelle en 43.6% in opslaanhutte. Slegs 12.1% volwassenes het minder as 20% van hulle lewe in ’n stedelike gebied spandeer. Ses-en-dertig is onder die armste tertiel van die bate indeks geklassifiseer. Die persentasie volwassenes met ’n LMI groter as 30 kg/m^2 was 63.3% vir vroue en 12% vir mans. Mans het ’n middelomtrek en MHV van 85.7 cm en 0.89 en vroue 97.5 cm en 0.85, onderskeidelik gehad.

Analise van die 24-uur dieetopname het baie lae gemiddelde energie innames getoon (M 6516 (2929; V 5760 (2446) kJ. Hierdie resultate het nie die hoë voorkoms van vetsug, veral in vroue, asook in vier ander studies onder stedelike swart bevolkings ondersteun nie. Gevolglik is die Goldberg vergelyking gebruik om onderrapporteurs uit te skakel. Die oorblywende steekproef het

544 (214 mans en 330 vroue) deelnemers behels wat te Khayelitsha (42.4%), Langa (31.4%), en Gugulethu (15.3%) woonagtig was, met minder as 10% te Crossroads en Nyanga.

Na die uitskakeling van onderrapporteurs was die gemiddelde energie-innames soortgelyk aan dié van die 1990 studie, en in 25–44-jarige mans was dit 8557 kJ vergeleke met 8500 kJ in 1990. In vroue was dit 7619 kJ vergeleke met 6400 kJ in 1990. Vetinnames was die hoogste in die 25–44-jarige mans (32% energie [E]) en vroue (33.4% E) in 2009 vergeleke met 1990 (mans: 25.9% E, vroue: 27.0% E). Koolhidraatinnames was laer in 2009 (mans 53.2% E, vroue: 55.5% E) as in 1990 (mans: 61.3% E; vroue: 62% E), terwyl suikerinname in vroue betekenisvol ($p < 0.01$) toegeneem het. Daar was betekenisvolle positiewe korrelasies tussen verstedeliking en totale vet ($p=0.016$), versadigde vet ($p=0.001$), mono-onversadigde vet ($p=0.002$) en vet as ’n %E inname ($p=0.046$). Verstedeliking was omgekeerd geassosieer met die inname van koolhidraat %E ($p < 0.001$). Oor die algemeen het mikronutriëntinnames betekenisvol verbeter vergeleke met 1990 met die uitsondering van kalsiuminname. Hierdie is moontlik te wyte aan die nasionale fortifisering van mielies en volgraanmeel. Van belang is dat energie en makronutriëntinnames almal betekenisvol was, asook duur van verstedeliking, volgens ’n lineêre regressiemodel met behulp van die gemiddelde toereikendheidsverhouding.

Die hoër vet en laer koolhidraat %E innames in hierdie bevolking demonstreer ’n oorgang na ’n meer verstedelikte dieet oor die twee dekades. Voorwaartse regressie is gedoen om betekenisvolle veranderlikes te identifiseer wat daarna in die lineêre regressiemodel ingesleutel is. In mans is LMI betekenisvol geassosieer met proteïeninname, terwyl in vroue LMI verband gehou het met versadigde vet, versadigde vet %E, koolhidraat, en koolhidraat %E. Ten opsigte van die duur van verstedeliking kan dit gemeld word dat koolhidraat %E totale vet %E energie, kalsium, dierproteïen en totale cholesterol betekenisvol geassosieer is met groter verstedeliking.

In die 2009 studie is gevind dat melkprodukte, die vleisgroep, peulgewasse, versadigde vet en harde (volvet) margarien in mans en vroue statisties laer was as dié in die 1990 studie. Hierdie is ook vir die graangroep gevind, maar slegs vir mans. Ten spyte hiervan het die 2009 studie vir die mans en vroue betekenisvolle hoër innames getoon vir eiers, vitamien C-ryke vrugte en groente en poli-onversadigde vetsuurbronne vergeleke met die 1990 groep. Hierdie is ook vir die graangroep gevind vir vroue. Die persentasie verbruikers van rooivleis, witvleis, eiers, groente en vrugte, en graan het van 1990 tot 2009 toegeneem. Vir die suiwel- en vetgroepe het die persentasie verbruikers afgeneem.

Gevolgtrekking: Die nutriëntinnames demonstreer dat terwyl sekere veranderinge tussen 1990 en 2009 plaasgevind het, die dieetpatroon aangaande voedselinname swak bly. Die dieet het meer verstedlik en aterogenies geword met betrekking tot vetverspreiding en koolhidraatinname, terwyl die verbruik van sekere voedselgroepe laag gebly het, soos die swak inname van suiwelprodukte en lae inname van vrugte en groente. Die algehele gemiddelde mikronutriëntinnames het egter toegeneem tot bokant die dieetverwysingsinnames, met die uitsondering van kalsiuminname.

Aanbeveling: Intervensies is dringend nodig om die verskuiwing na ’n voortgesette aterogeniese dieet teen te werk en die verbruik van voorkeurvoedselgroepe, soos suiwelprodukte en vrugte en groente te bevorder.

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LIST OF ACRONYMS AND ABBREVIATIONS

ADA:	American Diabetic Association
AHA:	American Heart Association
ALA:	α-linolenic acid
AMDR:	Acceptable macronutrient distribution ranges
AI:	Adequate intakes
AS:	Added sugar
BF%:	Body fat percent/percentage
BP:	Blood pressure
BMI:	Body mass index
BRISK:	Black Risk Factor study
CAD:	Coronary artery disease
CDC:	Centers for Disease Control
CHD:	Coronary heart disease
CHO:	Carbohydrates
CI:	Confidence interval
CORIS:	Coronary Risk Factor study
CRIBSA:	Cardiovascular risk in black South Africans
CRISIC:	Coronary Risk Factor study in Coloureds
CVD:	Cardiovascular disease
DALYs:	Disability-adjusted life years (DALYs)
DBP:	Diastolic blood pressure
DRI:	Dietary reference intakes
EAR:	Estimated average requirements
EER:	Estimated energy requirement
EI:	Energy intake
FAO:	Food and Agriculture Organization
GDP	Gross domestic product

HBV:	Hepatitis B virus
HCV:	Hepatitis C virus
HDL-C:	High-density lipoprotein cholesterol
HPV:	Human Papilloma virus
HC:	Hip circumference
IDF:	International Diabetes Federation
IGT:	Impaired glucose tolerance
LA:	Linoleic acid
LDL-C:	Low-density lipoprotein cholesterol
LMIC:	Low- and medium-income countries
MAR:	Mean adequacy ratio
MUFA:	Monounsaturated fatty acids
NCD:	Non-communicable diseases
Na:	Sodium
NAR:	Nutrient adequacy ratio
NFCS:	National Food Consumption Survey
OR:	Odds ratio
PA:	Physical activity
PAEE:	Physical activity energy expenditure
P:S ratio:	Polyunsaturated fatty acids to saturated fat ratio
PUFA:	Polyunsaturated fatty acids
PURE:	Prospective Urban and Rural Epidemiology study
QFFQ:	Quantified food frequency questionnaire
RDA:	Recommended dietary allowance
SA:	South Africa
SANHANES:	South African Health and Nutrition Examination Survey
SASPI:	South African Stroke Prevention Initiative
SBP:	Systolic blood pressure
SES:	Socio-economic status

SF:	Saturated fat
SFA:	Saturated fatty acids
SOC:	Sense of coherence
SSA:	Sub-Saharan Africa
TC:	Total cholesterol
TG:	Triglycerides
THUSA:	Transition, Health and Urbanisation in SA
TOD:	Target end organ damage
TS:	Total sugar
TUIL:	Tolerable upper intake level
VIGHOR:	Vanderbijlpark Information Project on Health, Obesity & Risk
WC:	Waist circumference
WHO:	World Health Organization
WHR:	Waist to hip ratio
YLD:	Years of life disability
YLL:	Years of life lost
%E:	Percentage energy
24HR:	24-hour recall

CHAPTER 1: INTRODUCTION

1.1 MOTIVATION FOR THE STUDY

The National Burden of Disease study (2000) highlighted that in addition to the substantial impact of HIV/AIDS as a cause of death in South Africa (SA), there is also a major health transition under way in terms of the growing burden of chronic non-communicable diseases (NCD).[1] As countries become more developed, socially and economically, their disease profile changes from one of infectious diseases, high infant and child mortality rate, and malnutrition, to a predominance of the degenerative, chronic diseases.[2] However, developing countries may also experience a double burden resulting from the simultaneous occurrence of both these disease spectrums, hence over- and under-nutrition can occur in the same community and even in the same household.[3,4] In fact South Africa is considered to have a triple-burden due to the high prevalence of HIV/AIDS found.[1]

Global trends and evidence from other countries undergoing an epidemiological transition predict that the prevalence of NCD, such as cardiovascular disease (CVD), type 2 diabetes mellitus and certain cancers have increased in the past few decades and is still rising.[2] In the 2001 World Health Organization (WHO) Bulletin, it was concluded that not addressing NCD would be irresponsible and unethical.[5] A research agenda for sub-Saharan Africa (SSA) that includes NCD would support appropriate programmes of preventative and clinical interventions within the context of the overall disease burden and the continuing pre-eminence of communicable diseases.[5] In 2014, the WHO estimated that 43% of total deaths in SA were caused by NCD and the probability of dying from the four main NCD (CVD, cancer, chronic respiratory diseases and diabetes) between ages 30 and 70 years, was 27%.[6]

In SA, a similar scenario has been taking place, although there is a paucity of data on the urban black population. In the South African Health Review of 2008 it was noted that ischaemic heart disease (IHD), stroke, hypertensive disease and diabetes mellitus were among the top causes of mortality for SA overall.[7] In the South African Health and Nutrition Examination Survey (SANHANES) that was undertaken in 2012, 10.4% of participants (over 15 years) were found to be pre-hypertensive, while 10.2% were hypertensive (≥140/90 mmHg).[8] Almost one out of five participants (18.4%) had impaired glucose levels and diabetes was diagnosed in 9.5% of adults.[8] Furthermore, 23.9% had abnormally high serum cholesterol, 28.8% had high low-density lipoprotein cholesterol (LDL-C) levels and 47.9% had abnormally low high-density lipoprotein cholesterol (HDL-C) levels. The prevalence of overweight and obesity was 19.6% and 11.6%, respectively, in males and 25% and 41% in females.[8]

Ischaemic heart disease was the largest single cause of death in the Western Cape population, accounting for 12% of all deaths in 2000.[1] In addition to IHD, stroke, chronic obstructive pulmonary disease, diabetes mellitus, and lung cancer were the five leading single causes of death, accounting for more than 50% of deaths.[1]

According to the Southern African Stroke Prevention Initiative (SASPI) it has been shown that SA is facing the challenge of an emerging epidemic of vascular diseases. Therefore, research is required to establish the social determinants of these risk factors and interventions dealing with their prevention and management to reduce both individual and population risk, particularly in black males.[9]

Non-communicable diseases are known to be influenced by modifiable risk factors, i.e. unhealthy diet, inactivity, tobacco use and irresponsible drinking of alcohol (Figure 1.1).[10] This, in turn, leads to intermediate risk factors like obesity and raised blood pressure (BP), raised blood glucose and/or abnormal lipid levels. The last published study with regard to the status of the modifiable risk factors for NCD in the African population in the city of Cape Town – the Black Risk Factor study (BRISK), took place in 1990, which was almost twenty years ago.[11] From these results, Steyn et al. (1991)[11] concluded that those who had spent larger proportions of their lives in an urban setting (i.e., Cape Town) tended to have unhealthier lifestyles. They were also at higher risk for developing NCD compared to their less urbanised counterparts.[11] One of the recommendations of the BRISK study was that NCDs could still be prevented in the black population, but preventative measures had to be rapidly instituted for all population groups. The researchers emphasised that the prevention of these degenerative diseases should be incorporated into evolving primary healthcare services.[11]

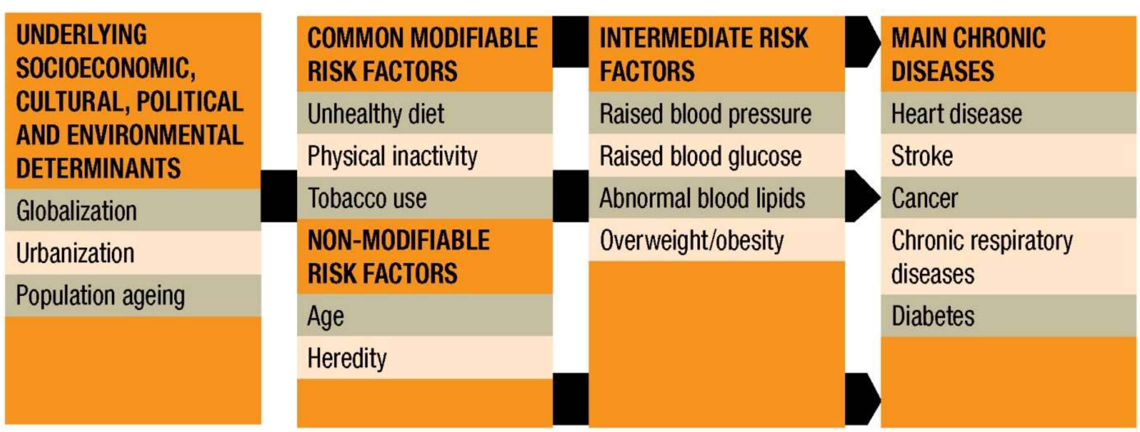


Figure 1.1: Common modifiable risk factors for the main non-communicable diseases[10]

More recent evidence has revealed that stunting in preschool children can co-exist with emerging obesity and other risk factors for CVD in the adult population.[12,13] This is not just a South African problem, since Popkin et al. (2001)[14] found that in China and India the same phenomenon exists. Under-nutrition is rapidly decreasing in both countries, while the proportion of overweight is increasing. This is accompanied by increases in hypertension, stroke, heart disease and type 2 diabetes. A similar scenario is also found in other African countries.[15,16]

In 2009, the cardiovascular risk in black South Africans (CRIBSA) study was undertaken in the same areas where a study was undertaken in 1990 (BRISK) to determine the prevalence of NCDs and the occurrence of associated lifestyle risk factors in the adult black population of Cape Town. The 2009 study was a large study once again looking at NCDs (diabetes and heart disease) and their risk factors. This thesis reports specifically on the dietary intake and anthropometric status of this population to ascertain whether dietary intake had changed since 1990. The study population included all black Africans aged 15 to 74 years living in the same townships as in the 1990 study. The population are mainly Xhosa speaking and many have migrated from the Eastern Cape Province rural areas to the city of Cape Town, often in search of jobs and a better life. The townships include houses, hostels and informal shacks found on the northern side of Cape Town.

CHAPTER 2: LITERATURE REVIEW

Since the focus of this study was on dietary intake and weight status the literature review covers the following facets identified in Figure 1.1, namely the common modifiable risk factors, and the immediate risk factors associated with the development of NCDs.

2.1 MODIFIABLE RISK FACTORS

2.1.1 DIET

To maintain a healthy dietary intake, energy and macronutrients should be consumed within certain parameters.[17] Energy intake (EI) should be in accordance with daily output in terms of metabolism and physical activity (PA) level.[17] The acceptable macronutrient distribution ranges (AMDR) are 45–65% of energy (E) intake for carbohydrates (CHO); total fat 25–35%E and protein 10–35%E to maintain a healthy balanced diet. Furthermore, a fibre intake of 38 g/day for adult males and 25 g/day is recommended for adult females, while it is recommended that added sugar (AS) intake should not exceed 10%E.[17] In addition to achieving healthy macronutrient intake levels, it is also essential that minerals and vitamins are consumed in amounts required for optimal health. In this regard, the following micronutrients are discussed throughout the thesis, since estimated average requirements (EAR) have been established.[17] Deficiencies of calcium, iron, zinc, B vitamins (B1, B2, B6, B12, niacin), vitamin A, folate, and vitamin C have been found to occur in South Africa, as documented in studies.[17]

A high salt intake and obesity are known to contribute to the development of raised BP and hypertension. The overall salt intake in SA is also known to be greater than that which is currently recommended, i.e. less than 5 mg/day. Seedat et al. (1996)[18] suggested that black African adults have an abnormal transport mechanism of sodium (Na) and rennin activity, thereby increasing their risk for hypertension. A high Na intake is common in SA, particularly in poor settings, as it is used to preserve food or to make food tastier. Food items high in Na, such as flavour enhancers (e.g. Aromat and soup powders) are added to many dishes, as they create a meaty taste, thereby increasing Na intake considerably.[18]

According to Popkin et al. (2001)[19] changes in diet and PA patterns are fuelling the obesity epidemic and these rapid changes within transitional societies are related to socio-economic and demographic changes. However, it is acknowledged that numerous other factors are also involved in the burgeoning obesity epidemic.

2.1.1.1 The nutrition transition

While it is acknowledged that physical inactivity, and irresponsible alcohol and tobacco use also play a role in the development of NCD, in this study we mainly focus on dietary intake. Since an unhealthy diet leads to changes in nutritional status, overweight/obesity, hyperlipidaemia, elevated glucose and elevated BP will also be described in terms of dietary factors.

Poor nutritional status is frequently the result of urbanisation and the associated nutrition transition. A diet is regarded as being an unhealthy one when it comprises the following element/s: high in energy (kJ); high in total fat, saturated and trans fatty acids; high in Na; high in refined CHO and AS, and low in fibre and intake of fruit and vegetables.[10,20]

The nutrition transition refers to changes in dietary patterns and nutrient intakes of individuals, families, groups of people, or whole populations when their food environment and other circumstances change.[21] These changes are frequently associated with urbanisation.[13,22] The nutrition transition is usually accompanied by a demographic transition (a shift from a pattern of high fertility and high mortality to one of low fertility and low mortality) as well as an epidemiological transition (a shift from a pattern of high prevalence of malnutrition-related infectious diseases to a pattern of increases in the nutrition-related, degenerative, chronic diseases of lifestyle).[21] Furthermore, it is characterised by the population shifting from eating a diet high in CHO and fibre and low in fat to eating a diet lower in CHO, high in fat (particularly saturated and trans fatty acids) and low in fibre.[22] To fully understand the nutrition transition, it is important to understand the changes associated with this transition (Figure 2.1).

There are societal, technological and political factors which influence the nutrition transition (Figure 2.1). Globalisation of markets influences nutrition patterns, for example, more take-away establishments are available in the cities and the media also plays an important role in influencing people to move from a traditional diet to the typical westernised diet. The world is becoming smaller because of modern technology and globalisation of markets, economies, and multinational companies having collectively created a food environment in which a huge variety of good tasting, palatable, “healthy” and “unhealthy” foods and products are available during all seasons.[21] This is particularly relevant in urbanised areas.[22] The food industry is also a strong role player which operates for profit. The third party are the policymakers such as UN agencies and government who make policy regarding food and health. In addition the food environment also influences the nutrition transition as do individual and family members.

Societal, Technological and Political Factors	Changes during the Nutrition Transition	Individual, family factors
• Globalisation Trade Urbanisation Agriculture Media • Governance Food and nutrition policy and service Economics Poverty Food security • Food Industry “New” products Profit motive Health agenda • UN Agencies, NGOs Recommendations for policy Intervention programmes	Food Environment: What is available at what prices ↓ Food behaviours, eating patterns (food choices) ↓ Nutrient intakes ↓ Biological response to diet ↓ Nutritional status Risk factors for chronic diseases of lifestyle ↓ Morbidity, Mortality Food behaviours, eating patterns (food choices) ↓ Nutrient intakes ↓ Biological response to diet ↓ Nutritional status Risk factors for chronic diseases of lifestyle ↓ Morbidity, Mortality	• Household and individual food security/Insecurity, Income(poverty), Education, Socio-economic position, Knowledge, Culture, Peer-pressure • Psychological strengths, • Behaviour • Genetics, • Biological programming

Figure 2.1: A conceptual framework to illustrate the factors influencing the causes and consequences of the nutrition transition[21]

Popkin et al. (2011)[23] report that the world has experienced a marked shift in the global body mass index (BMI) distribution towards reduced under-nutrition and increased obesity. Modern technology, globalisation, government policies and food industry practices have created an energy imbalance in terms of the number of calories people consume. Increased frequency of meals and increased portion sizes are examples of this transition. Therefore, it is important to understand the causes of changes in the key aspects of our diets, i.e. by not just the role of beverages, but also various components of our diet with regards to energy imbalance.[14,19] Research is critical if we are to establish a balance between energy intake and expenditure, while at the same time creating a healthier dietary pattern not just for health, but also to prevent disease.

Similarly, the ongoing nutrition transition and the associated dietary changes have been reviewed by Belahsen et al. (2014)[24] in low- and middle-income countries (LMIC). They reported that the nutrition transition is accompanied by demographic and epidemiological transitions which are associated with economic development and urbanisation.[24] In these countries, they found that while problems of hunger and under-nourishment persist, there is also an escalation of diet-related NCD making them face problems of malnutrition, under- and over-nutrition, in addition to the high prevalence of infectious diseases still found in many LMIC.[24]

We know that in addition to protein-energy malnutrition, underweight and micronutrient deficiencies affect a high proportion of children and females. However, changes in dietary habits and PA patterns have led to the emergence of chronic NCDs and their risk factors, such as obesity, diabetes, hypertension, stroke, hyperlipidaemia, coronary heart disease (CHD) and cancers.[2] One possible reason for an increase in obesity and its associated health consequences, is the increase in the consumption of energy-rich ready prepared meals and the continuous emergence of fast-food restaurants leading to a high consumption of foods rich in sugar and fat.[19] Another finding is that the health problems associated with the nutrition transition have not spared populations in the Mediterranean area, who reportedly follow a healthier diet which is protective against CVD risks.

2.1.1.2 Diet and the nutrition transition in South Africa

To better understand dietary factors, a history of studies undertaken in SA, reflecting the nutrition transition, will be described by starting with the earliest and concluding with the most recent ones presented by population group.

2.1.1.2.1 White population

A dietary survey done in 1979 on a randomly selected sample of 15% of the 1979 Coronary Risk Factor study (CORIS) in the white population, showed that this population consumed a typical Western diet.[25] Additional analyses were then performed to identify specific weaknesses in the dietary habits of this population. They found that the meat group was the main source of total fat, saturated fat (SF) and dietary cholesterol intake. The fat group was the second most important source of total fat in the diet, while the milk group was the second most important source of SF. Furthermore, this study population preferred refined cereals and had a low fruit and vegetable intake. This athrogenic diet was thought to play a large role in the adverse CHD risk profile found in the CORIS population.[25]

Twenty years later in 1988, a study describing the nutrient intakes of a group of randomly selected white males and females participating in the Vanderbijlpark Information Project on Health, Obesity and Risk Factors (VIGHOR) was done.[26] They showed that not much had changed since 1979 as the mean intakes of the different age groups of males and females did not meet prudent guidelines regarding fat and fibre intakes. Also, the mean intake of protein, vitamin E, vitamin B12 and phosphorous were adequate in the 35–55-year-old group, but their intake of vitamin D, folate, calcium, magnesium, selenium, potassium, copper and iron appeared to be inadequate. This would place them at risk for developing diseases of both over- and under-nutrition. A simple focused dietary message was recommended to be promoted among this community to increase their fruit, vegetable, wholegrain and legume consumption in order to rectify observed nutritional problems.[26]

2.1.1.2.2 Coloured population

Similarly, the Coronary Risk Factor Study in Coloureds (CRISIC) was conducted in 1982 as part of a cross-sectional survey of the prevalence of CHD risk factors among the so-called coloured population (mixed ancestry) of the Cape Peninsula.[27] A specific meal pattern seemed to emerge in this population: very little or no breakfast, lunch consisting mainly of bread, a cooked supper, and heavy snacking between meals. Despite the fact that these snacks frequently comprised basic foods (sandwiches with meat balls, Vienna sausages, polony, eggs or cheese, or commercially fried fish and chips), it did not compensate for an inadequate breakfast. Snacks also tended to supply large quantities of sugar and fat together with the more nutrient-rich foods. Intake from the milk group was inadequate, and more than the recommended number of portions from the meat group were eaten. Vegetable and fruit consumption was also low. Thus, this population had an eating pattern that could contribute to the development of NCD given their low-fibre and high-fat intake levels, but in addition to diseases of malnutrition such as osteoporosis and some vitamin deficiencies.[27]

2.1.1.2.3 Indian population

In a study, investigating the dietary intake of the Indian population in Durban, researchers reported on the energy and nutrient intakes as risk factors for CHD in the nineties.[28] They found that the energy intake tended to decrease with an increase in age in males and females. In males, the median energy intake was 67% or less of the recommended dietary allowance (RDA), while in females, the median energy intake was approximately 60% of the RDA. Furthermore, in the various age groups animal protein accounted for 46.3–63.7% of total protein intake, compared to the recommended maximum of 50%. They also reported that the percentage of energy derived from total fat varied between 32.3% and 34.9% in males and between 33.1% and 36.1% in females, which was also higher than the recommended 25–30% at that time. They found that the intake of polyunsaturated fatty acids (PUFA) was high, more than 10% E, with a median PUFA to SF ratio (P:S ratio) that varied between 1.38 and 1.96 in the different age and sex groups. The P:S ratio was determined as being high and its effect on the oxidation of low-density lipoprotein cholesterol (LDL-C) in this population, with a high prevalence of CHD, should be investigated as a possible risk factor for CHD. Dietary fibre intakes were also found to be low.[28]

In a later study, Naicker[29] assessed the association of dietary and lifestyle exposures with the risk of NCD among 250 apparently healthy Indian adults in KwaZulu-Natal. A quantitative food frequency questionnaire (QFFQ) validated by three 24-hour recalls (24HRs) was used. For the males, the total fat intake was 35.1% E and 37.1% E for the females, while the mean total protein intake was 12.8% E for the males and 12% E for the females. The mean total CHO intake as a percentage of total energy intake also fell within the dietary reference intakes (DRI) for both the males and females (49.9% E and 47% E, respectively). However, the mean total AS intake was found to be high, 59.6 g and 45.4 g for males and females, respectively. In addition, the mean fibre intake for both males (18.8 g) and females (18.1 g) was found to be lower than the RDA. Naicker[29] concluded there was a need to increase the consumption of fruits and vegetables, decrease fat intake, reduce BMI, and increase PA levels for a sustainable intervention strategy to reduce and control the burden of NCD in the Indian population. Thus, these studies showed a very similar situation to what was found in the white and Coloured population.

2.1.1.2.4 Black population

In the BRISK study,[34] in 1990, the dietary pattern of this urban black sample was found to meet the prudent dietary recommendations of the American Heart Association (AHA),[30] in terms of dietary fat intake. The AHA recommends between 20% to 35% of E should be derived from fat.[30] The mean total fat consumption in the BRISK study was 27% E, however, it already reflected a transitional phase towards the Western diet when compared to other studies of Africans in rural areas.[31] The Heart and Stroke Foundation recommend that not more than 1% of total fat should be derived from trans fatty acids and that SF should account for no more than 7% to 10% of total fat intake.[32]

Findings of the BRISK study further indicated that certain micronutrient intakes, commonly found in a variety of fruits and vegetables, were inadequate in this population.[12] Actual food intakes reflected a largely refined cereal-based diet, which was low in dairy products and in fruits and vegetables. The inadequate dairy consumption found in this study, presented a shift from tradition, as the rural African black population normally consumes higher quantities of milk, especially sour milk or maas.[12] In addition, the intake of meat and meat products was found to be threefold higher than that of rural African blacks.[12] The intake of cereal and cereal products by urban participants far exceeded that of rural African blacks, although the latter had a higher CHO intake. This is, therefore, further evidence of a transition towards a more westernised diet.

Stratification of fat intake in terms of duration of urban exposure also revealed a trend of increasing fat intake with longer duration of urban exposure.[12] Steyn et al. (1998)[33] proposed that a proportion of this community (BRISK study) may have been nutritionally deprived during childhood, and had moved from under-nutrition to extreme over-nutrition without having achieved optimal nutritional status.

Furthermore, in the BRISK study, the black urban population demonstrated an adoption of the Western diet. Their CHO was <65% E, fibre intake was lower than recommended, and fat intake was higher (>25% E).[34] Also, changes in intake of different types of food and food groups over time were observed. There was a higher consumption from the following food groups: meat, fruit and vegetables, fats and non-basic foods (such as drinks and sweets), while there was a decreased consumption from the dairy and cereal groups. There was also a larger consumption of sugar-containing food items in urban versus rural areas.

In Limpopo, the dietary intake, weight status and nutrition knowledge of young black African females attending the University of Limpopo (Sovenga campus) was evaluated in the late nineties.[35] This study identified whether students came from urban or rural areas. Rural students were found to follow a more traditional African diet consuming more cereals (maize meal) and legumes, and less confectionary and beverages compared to their urban counterparts. Macro- and micronutrient intakes were similar in both groups with calcium, iron and zinc intakes being least optimal.

The effects of urbanisation on nutrition and health in the North West Province of SA were also assessed in the study on Transition, Health and Urbanisation in SA (THUSA).[37] The researchers stratified their subjects into five groups representing different levels of urbanisation, viz. deep rural, farms, informal settlements, townships and towns/cities. When they compared females from the most rural group (1) with the most urban group (5), the following dietary trends were observed: %E from CHO, 67.4% vs. 57.3%; from fats, 23.6%E vs. 31.8% E; from animal protein, 22.4 g vs. 42.6 g per day, respectively. Furthermore, females in groups 1 to 5 had overweight and obesity (BMI ≥25) rates of 48%, 53%, 47%, 61% and 61%, respectively, showing an increase in weight in accordance with the increase of urbanisation. Thus, it could be deduced that with progressive westernisation and improvement in socio-economic status (SES), the diet tends to become unhealthier due to an increase in animal protein and fat and a decrease in CHO. Consequently, a higher fat intake, particularly SF intake, together with a high Na intake as well as a reduction in fibre intake will increase the risk of developing NCDs.

Vorster[22] also highlighted the fact that mortality rates from CVD confirmed that stroke is a major public health problem among black South Africans, possibly because of an increase in hypertension, obesity and smoking during various stages of urbanisation. Therefore, a reduction in the contributing risk factors, particularly among urbanised black South Africans is of paramount importance as it directly affects mortality rates.

In 2008, a cross-sectional study was done in Bloemfontein (urban area) to determine the macronutrient intake of adult black African females.[38] Hattingh et al. (2010) [38] found that the median total energy, protein and CHO intakes of all females exceeded the DRI. The median intakes of fibre were low, while median fat intakes were high. Younger females had significantly higher intakes of total fat (p=0.034), SF (p=0.046) and PUFA (p=0.015). Median energy distribution was 12% protein (both age groups), 32% fat (younger females) and 31% fat (older females) and 51% CHO (younger females) and 53% CHO (older females). They concluded that the high median energy and macronutrient intakes may pose a potential risk for the development of NCDs. Also, the diverse eating pattern followed by these females has led to the consumption of an energy-dense diet, high in proteins and fats. However, many of these foods were consumed in the refined form and could be considered as one of the major factors that contributed to the inadequate fibre intake. The high total fat intake is likely by reason of the dietary inclusion of fatty animal foods, hydrogenated fats and vegetable oils, and the selection of fried snacks and convenience foods.

The Prospective Urban and Rural Epidemiology (PURE) study determined the occurrence of lifestyle risk factors associated with NCD in black adults in North West (unpublished data from Wentzel-Viljoen). In particular, this specific study focused on the dietary intake and nutritional status of this population to ascertain whether dietary patterns/habits have changed in urbanised Africans in the North West Province. The mean energy and macronutrient intakes were found to be considerably higher in the urban areas. In these areas, energy intakes were 15 485 kJ for males and 12 302 kJ for females, while in rural areas these were 10 084 kJ for males and 9891 for females. The mean protein intakes for males and females in urban areas were 119 g and 97 g per day, respectively, and for rural males and females 72 g and 69 g per day, respectively. In urban areas, the mean fat intake for males was 119 g and 72 g per day in rural areas, while for females this was 63 g and 66 g per day in urban and rural areas, respectively. Thus, urban participants consumed a diet high in energy and fat and low in fibre.

Pretorius et al. (2012)[39] described the dietary intake and potential nutritional deficiencies in black African patients diagnosed with heart failure. Dietary intake in 50 consecutively consenting heart failure patients (mean age: 47 ± 18 years, 54% female) attending a major hospital in Soweto, were surveyed using a validated QFFQ. Food intakes, translated into nutrient data were compared to recommended values. In females, food choices likely to negatively impact on heart health included AS [consumed by 75%: median daily intake (interquartile range) 16 g (10–20)], sweet drinks [54%: 310 ml (85–400)], and salted snacks [61%: 15 g (2–17)]. Corresponding figures for males were AS [74%: 15 g (10–15)], sweet drinks [65%: 439 ml (71–670)], and salted snacks [74%: 15 g (4–22)]. The females' intake of calcium, vitamin C, and vitamin E was only 66%, 37% and 40% of the age-specific requirement, respectively, whereas, equivalent figures for males were 66%, 87% and 67%, respectively. Mean Na intake was 2 372 g/day for males and 1 972 g/day for females, i.e. 470% and 294%, respectively, of the recommended consumption levels. These researchers concluded that the nutritional status of black African patients with heart failure could be improved by recommending healthier food choices and reducing the intake of sweet drinks and excessive salt.[39]

Similarly, MacIntrye et al. (2012)[40] investigated the absolute micronutrient intake and the possibility of micronutrient dilution of AS in the diets of an African population in nutritional transition. This study was part of the THUSA study[37] mentioned earlier. The outcome measures were the macronutrient and micronutrient intakes of subjects in different quartiles of AS intake and BMI. They found that the average intake of AS was 10.01% E (67.12 g) in males and 11.2% E (67.10 g) in females compared to the recommended intake of 10%E. Respondents who consumed the most AS had significantly lower mean intakes of alcohol, but higher intakes of energy, macronutrients and most micronutrients. The diets of those in the highest sugar-intake group contained significantly less thiamine, riboflavin, niacin, vitamin B12, pantothenic acid, biotin, magnesium, phosphorus and zinc per 4.18 MJ. At every level of AS consumption, the mean intakes of fibre (males only), folate, ascorbic acid and calcium (males and females) did not meet the DRI [EAR]. Pantothenic acid and biotin (females only) did not meet the adequate intake. Respondents who consumed the most AS had significantly higher intakes of fruit (males only), bread and soft drinks, and lower intakes of maize meal and alcohol (males and females). MacIntrye et al. (2012)[40] concluded that the absolute intakes of most micronutrients were significantly higher in consumers with a high sugar intake compared to the lowest consumers of sugar.

Tydeman-Edwards[41] determined the diet and anthropometric status of adults 25–64 years old ~~and~~ pre-school children 0–7 years old in rural and urban areas of the Free State. For males, the total fat intake as a percentage of energy was found to be higher in the rural group (25.2%E) compared to the urban group (23.3%E), although this was not statistically significant. The intake of food groups and the frequent intake of certain food items (sugar, fats, fruit and vegetables) reflected the presence of a greater nutrition transition in the rural than urban communities included in the study. This is the only study in SA in which transition was higher in the rural group. The energy intake and macronutrient distribution in their diets were within prudent dietary guidelines.

Dietary intake, as reflected by the intake of adequate numbers of foods from the different food groups, was found to be mostly inadequate and thus according to this reference, the nutrition transition was more pronounced in the rural population. Unlike other studies, rural adults and children consumed more than their daily recommended servings of fats and oils, sweets and sugar, and meat and meat products. They also ate less fruit and vegetables than their urban counterparts. Urban adults and children tended to consume above their daily recommended servings of bread and cereals more often than their rural counterparts. In most instances sugar was the most frequently consumed food item, especially among rural participants, and cooked porridge was the most frequently consumed starchy food, followed by bread.

Tydeman-Edwards[41] determined that fruit and vegetables were consumed frequently, but fruit more so than vegetables. However, these intakes were mostly below the recommended number of servings per day as evidenced by the food intake from the different food groups. Despite the relatively prudent energy intake and macronutrient distribution, the intake of foods from different food groups and the intake of specific foods did not fall within the recommendations for a prudent intake.

Hattingh et al. (2013)[42] investigated the sugar consumption of poor black females in Mangaung. The consumption of total sugar (TS) and AS was compared between older and younger females as well as in socio-demographic, anthropometric and biochemical (blood lipids, glucose, insulin) categories. They found that AS consumption was higher in younger females living in brick houses and those who possessed a microwave oven. In older females, consumption of AS was higher in husband-headed households. Underweight females, with the lowest BMI, had a higher sugar consumption than overweight and/or obese females. Those with a lower body fat percentage (BF%) had a higher consumption of AS than females with a high BF%. Sugar consumption was significantly lower in younger females with elevated serum lymphocyte counts. Total sugar and AS consumption was higher in younger females with elevated serum glucose levels. Older females with elevated serum insulin had a significantly higher TS consumption compared to those with normal insulin concentrations. Thus, the amounts of TS and AS consumed by females in this observational study were unlikely to contribute to overweight and/or obesity.

However, in a similar study done in Johannesburg using data from the longitudinal birth cohort study, the Birth-to-Twenty (Bt20) cohort started in 1989, 17 to 18-year-old young adults in Soweto were studied.[43] The availability and accessibility of fast foods, sugar-sweetened beverages and snacks in urban SA may be contributing to the burgeoning obesity epidemic in adolescence. Therefore, these researchers wanted to determine the consumption of purchased foods and drinks and estimate the AS and dietary Na intake from these foods and beverages. They found that respondents consumed on average three times the recommended daily intake of AS (not more than 25 g of sugar for females, and not more than 38 g for males), and more than half of the recommended daily salt intake from purchased foods alone. Feeley and Norris[43] concluded that the dietary patterns during adolescence may exacerbate the risk of obesity and hypertension in later adult life.

In another study, Vorster et al. (2014)[44] assessed the relationship between AS intake and NCD risk factors in an African cohort study. Added sugars were defined as all monosaccharides and disaccharides added to foods and beverages during processing, cooking, and at the table. They conducted a 5-year follow-up of a cohort of 2010 urban and rural males and females, age 30–70 years at recruitment in 2005, from the North West Province in SA. They found that AS intake, particularly in rural areas, had increased rapidly in the 5 years since the baseline study. In rural areas, the proportion of adults, who consumed sucrose-sweetened beverages, had approximately doubled (25% to 56% for males, and 33% to 63% for females) in the same 5-year period. After adjustment, subjects who consumed more AS [(more than 10% E compared to those who consumed less AS had a higher waist circumference (WC) mean difference (95% CI): 1.07 cm (0.35, 1.79 cm)] and BMI (in kg/m^2) [0.43 (0.12, 0.74)] and lower HDL-C [-0.08 mmol/L (-0.14, 0.002 mmol/L)]. They concluded that this cohort showed dramatic increases in AS and sucrose-sweetened beverage consumption in urban and rural areas, and that this increased consumption was associated with increased NCD risk factors. In addition, it was shown that the nutrition transition had reached a remote rural area in SA and that urgent action was needed to address these trends.

According to all these South African studies, it is clear that the population has undergone and is still undergoing nutrition transition. This is with detrimental effects in terms of weight status as well as the increase in the prevalence of the risk factors for NCD. Although this study only investigated the adult population, these trends are also seen in young children. The National Food Consumption Survey (NFCS), undertaken in children 1–9 years old in 1999, was the first national dietary study in SA. Trends consistent with a population undergoing nutrition transition were found, i.e. differences in diet between urban and rural children, with urban diets being higher in energy and fats, while rural children consumed more CHO.[45,46]

2.1.1.3 Diet and the nutrition transition in other developing countries

Mehio-Sibai et al.(2011)[48] examined the burden of CVD risk factors in the Middle East and North Africa by means of a systematic review on dietary risk factors. Dietary patterns were derived from the WHO Food and Agriculture Organization (FAO) Statistical Databases. They found that wide variations exist across countries regarding the prevalence of CVD risk factors, i.e. obesity, diabetes, hypertension, hyperlipidaemia, smoking and physical inactivity. Some of these countries showed high values of certain factors which approach those that were observed in the developed world.

Obesity prevalence rates have reached alarming levels, particularly among females in the oil-rich countries (over 40%) making it the most pressing health concern in the region. Trends in dietary patterns illustrated a consistent rise in total energy supply by approximately 730kcal(3051kJ) per capita per day between 1970 and 2005. In addition, dietary patterns showed an increased consumption of fat and animal protein and a decreased intake of CHO, particularly wholegrain cereals and fresh fruits and vegetables.[48]

Mehio-Sibai et al. (2011)[48] concluded that regional differences were attributed to differences in lifestyle, occupation and a shift from traditional food habits. Thus, our understanding of the CVD disparities across various geographic regions is key to our effort in planning relevant intervention programmes. They also recommended that public health efforts should focus on obesity, physical inactivity and unhealthy dietary practices.[48]

In a study done in Mozambique, in 2005, the fruit and vegetable consumption is described according to socio-demographic characteristics and place of residence (urban/rural).[49] A national representative sample (n=3323) of subjects, aged 25–64 years, was evaluated in 2005 following the WHO Stepwise Approach to Chronic Disease Risk Factor Surveillance, which included an assessment of usual fruit and vegetable consumption (frequency and quantity). Less than 5% of the subjects reported an intake of five or more daily servings of fruits/vegetables and that females more often consumed fruits and vegetables and also in rural settings.[49]

In urban areas, the prevalence of fruit intake increased with education from 2 servings per day to 6 servings per day, but not with income. Conversely, they found, vegetable consumption (2 servings per day) was less frequent in more educated urban males and more affluent rural females. Thus, this population also showing signs of a westernised diet low in fibre intake resulting from a low intake of fruit and vegetables.[49]

Mayén et al. (2014)[50] in a systematic review, assessed the relationship between SES and dietary intake in Brazil, China and Iran. They reviewed cohort and cross-sectional studies in adults published between 1996 and 2013. They found that high SES or living in urban areas was associated with higher intakes of calories, protein, total fat, cholesterol, PUFA, SF, and monounsaturated fatty acids (MUFA), iron, and vitamins A and C, with lower intakes of CHO and fibre. This is similar to what is found in SA.[50]

They also found that a high SES was associated with higher fruit and/or vegetable consumption, diet quality, and diversity. They concluded that in these countries, high SES or living in an urban area is associated with overall healthier dietary patterns. However, it is also related to higher energy, cholesterol, and SF intakes. Thus, a dietary pattern that reflects variety and diversity can also lead to increased intakes of unhealthy foods, and social inequalities in dietary intake should be considered in the prevention and control of NCD in LMIC.[50]

Zaghloul et al. (2013)[51] described nutrient intakes and prevalence of overweight and obesity in a nationally representative sample of Kuwaitis in order to compare intakes with reference values. This was in the form of a national nutrition survey covering all geographical areas of the country. The subjects (n=1704) were between 3 and 86 years old. They found that obesity was more prevalent among females than males (50% and 70% for females aged 19–50 years and >51 years, respectively, vs. 29% and 42% for their male counterparts). Boys were more obese than girls, with the highest obesity rate among those aged 9–13 years (37% and 24% of males and females, respectively).[51]

Energy intake was higher than the estimated energy requirements for almost half of Kuwaiti children and one-third of adults. The EAR was exceeded by 78–100% of the recommendation for protein and CHO. More than two-thirds of males aged >4 years exceeded the tolerable upper intake level (TUIL) for Na. Conversely, less than 20% of Kuwaitis, regardless of age, consumed 100% or more of the EAR for vitamins D and E, calcium, and n-3 and n-6 fatty acids. Less than 20% of the children met the recommended level for fibre.[51]

Zaghloul et al.[51] concluded that the nutrition in transition in Kuwaitis was demonstrated by the increased prevalence of obesity and overweight, increased intakes of energy and macronutrients and decreased intakes of fibre and micronutrients. This is also in line with studies done in SA.

Baker et al. (2014)[52] explored the role of processed foods and beverages in Asia. Processed foods are known to have a tendency to be high in nutrients associated with obesity and diet-related NCD, such as refined sugar, salt, saturated and trans-fats. They thus identified the most significant ‘product vectors’ for these nutrients and described changes in their consumption in a selection of Asian countries. They found that sugar, salt and fat consumption from processed foods has plateaued in high-income countries, but has rapidly increased in the LMIC. Also that relative to sugar and salt, fat consumption in the upper-middle- and LMIC is converging most rapidly with that of high-income countries. Carbonated soft drinks, baked goods, and oils and fats are the most significant vectors for sugar, salt and fat, respectively.[52]

According to these researchers, there appears to be convergence in consumption patterns of processed foods at the regional level, but country-level divergences including high levels of consumption of oils and fats in Malaysia, and soft drinks in the Philippines and Thailand. Thus, these countries all consume a high level of processed foods, however, different countries have differences in the highest consumption of particular processed foods. They suggested that policy-makers should take action to prevent or decrease processed food consumption as comprehensive policies are most likely to be effective in achieving these goals. This too is very similar to findings of those in SA.[52]

In a study in India, Bowen et al. (2011)[53] assessed the diets of migrants of rural origin, their rural dwelling siblings, and those of urban origin together with their urban dwelling siblings using a semi-QFFQ questionnaire. Migrant and urban participants reported up to 80% higher fruit and vegetable intake than rural participants, and up to 35% higher added sugar intake. Meat and dairy intake was higher in migrant and urban participants than rural participants, but varied by region. They concluded that rural to urban migration appears to be associated with both positive (higher fruit and vegetables intake) and negative (higher energy and fat intake) dietary changes and that these changes are of relevance to cardiovascular health and warrant public health interventions.[53]

Dietary shifts have been found to occur almost concurrently with demographic and epidemiologic shifts, urbanisation and industrialisation and together contribute to increased prevalence of nutrition-related NCD.[54] The emergence of the nutrition transition in Southern African countries was examined using anthropometric measures, NCD prevalence, and food consumption data. The findings reveal a growing prevalence of overweight and obesity across these countries with a national prevalence estimated between 30% and 60% in all but two Southern African countries. Overweight prevalence in excess of 60% has been reported in some sub-population groups.[54]

Hypertension prevalence of at least 30% has also been reported. Furthermore, the prevalence of overweight and obesity and hypertension in many Southern African countries exceeds that of HIV and is often on par with stunting in children. Non-communicable diseases are equally a serious public health problem as stunting and HIV. Collectively, nutrition-related NCD explain 20–31% of mortality for Botswana, SA, Swaziland, Mozambique and Zambia. At least 72% of adults in Southern African countries have fewer fruit and vegetable servings daily than recommended. Additionally, adults in Southern African countries do poorly in PA; 31–75% do not exercise regularly. Not surprisingly, in

Southern African countries, 15–40% of adults have at least three risk factors of CVD and are grappling with nutrition-related NCD which threaten to surpass the infectious diseases burden.[54]

Bosu et al. (2015)[55] reports that the nutrition landscape in West Africa has been dominated by programmes to address under-nutrition. However, with increasing urbanisation, technological developments and associated change in dietary patterns and PA, childhood and adult overweight and obesity are becoming more prevalent. There is an evidence of increasing intake of dietary energy, fat, sugars and animal protein, with a low consumption of fruit and vegetables, universally, in West Africa. Overall, the foods consumed are predominantly traditional, with the component major food groups within recommended levels.

Most of the West African countries are at the early stages of nutrition transition, but countries such as Cape Verde, Ghana and Senegal are at the latter stages. In the major cities of the region, children consume energy-dense foods such as candies, ice cream and sweetened beverages up to seven times as frequently as fruit and vegetables. Adult obesity rates have increased by 115% in 15 years since 2004. In Ghana, the prevalence of overweight/obesity in females has increased from 12.8% in 1993 to 29.9% in 2008. In Accra, overweight/obesity in females has increased from 62.2% in 2003 to 64.9% in 2009. The age-standardised proportion of adults who engage in adequate levels of PA ranges from 46.8% in Mali to 94.7% in Benin. The lingering stunting in children and the rising overweight in adults have resulted in a dual burden of malnutrition affecting 16.2% of mother-child pairs in Cotonou.[55]

The prevalence of hypertension has been increased and ranges from 17.6% in Burkina Faso to 38.7% in Cape Verde. This prevalence is higher in the cities, e.g. 40.2% in Ougadougou, 46.0% in St Louis and 54.6% in Accra. Diabetes prevalence ranges from 2.5% to 7.9%, but could be as high as 17.9% in Dakar, Senegal. The consequences of the nutrition transition are not only being felt by the persons with high SES, but also in cities such as Accra and Ouagadougou, where at least 19% of adults from the poorest households are overweight and 19–28% have hypertension.[55]

Akarolo et al. (2013)[56] conducted a cross-sectional study in Nigeria to determine dietary intake and weight status. The main CHO food eaten was rice (48.6%) followed by fufu (roots and tubers) (30.5%) and bread (13.1%). The prevalence of overweight and obesity in females, was 63% and 73%, respectively, compared to 56% of males. Parboiled long grain white rice was shown to be the most commonly consumed CHO by urbanised Nigerians. Other traditional CHO foods are still consumed frequently and remain quite popular.[56]

Delisle[57] identified dietary patterns that can be recommended in health promotion endeavours, particularly in population groups undergoing the nutrition transition. These researchers also identified healthy and culturally relevant dietary patterns that can be promoted as a means of preventing diet-related chronic diseases. They examined the dietary intake of population groups of African origin living in Canada (Montreal), Europe (Madrid), and West Africa (urban and rural Benin). Their findings indicated that a limited number of foods predicted diet quality and health-related outcomes in various population groups. Fruit and vegetables, fish, wholegrain cereal, and legumes, in particular, are foods on the protective side, whereas sweets, processed meats, fried foods, fats and oils, and salty snacks are on the negative side.[57]

They recommended that further research on dietary patterns and their healthfulness is required in diverse food cultures. Furthermore, they suggested that in groups of African origin, traditional diets are healthier than the non-traditional dietary patterns that have evolved with globalization, urbanisation, or acculturation, although micronutrient intakes need to improve. Additionally, healthy eating patterns are only feasible if access to food is adequate.[57]

An investigation to whether variability in food availability for consumption, lifestyle and socio-demographic factors are associated with the worldwide prevalence of overweight, obesity and hypertension was done by Siervo et al. (2014)[58] in an ecological analysis. They obtained country-specific prevalence estimates of overweight, obesity and hypertension. Prevalence estimates were then matched to year and country-specific food and energy availability for consumption of cereals, sugar, sweeteners and honey, vegetable oils, fruits, starchy roots, pulses, total vegetables, alcoholic beverages, total meat, animal fat, eggs, milk, and fish and seafood. The per capita gross domestic product (GDP), urbanisation rates, and prevalence of physical inactivity for each country were also obtained. The study investigators evaluated databases with information on overweight, obese and hypertension statistics from 128, 123 and 79 countries, respectively. They found that consumption of sugar and animal products were directly associated with GDP and urbanisation rates.[58]

In a multivariate regression model, physical inactivity, cereal consumption, and sugar consumption were significant predictors of obesity prevalence. Midpoint age, prevalence of overweight and consumption of cereals were significant predictors of hypertension. Their analyses also showed that the prevalence of obesity in females was greater than in males. They concluded that high sugar consumption and sedentary lifestyle are associated with increased obesity prevalence. The non-linear association of sugar consumption, with prevalence of obesity, suggests that effective strategies to reduce its consumption may have differential effects in countries at different stages of ~~the~~ nutrition transition.[58]

Geddam et al. (2015)[59] explored the risk factors for CVD, hypertension and type 2 diabetes among tribal migrants living in urban areas of India. A population-based cross-sectional study was carried out on tribal migrants, aged ≥30 years (n=138 males, n=137 females) who were of low SES, living in an urban slum area (Kondapur) of Hyderabad. Blood lipids, glucose, homocysteine, glycated haemoglobin (HbA1c), BP and nutritional biochemical markers such as serum albumin, serum protein, vitamin D and haemoglobin were examined. The prevalence of overweight in males and females was 35.3% and 32.4%, respectively, while general obesity was 14.3% and 24.3%, respectively. In addition, high concentrations of total cholesterol (TC), LDL-C, triglycerides (TG), homocysteine and HbA1c in the study population was also observed. Duration of stay in urban areas had no significant association with overweight and obesity.[59]

Most tribal migrants did not meet at least 50% of RDA of micronutrients such as iron (80–84%), vitamin A (81–83%) and riboflavin (67–84%). A similar finding was observed with food groups such as leafy vegetables (84–91%), and milk and milk products. However, the consumption of fat and protein was found to be ≥70% indicating a transition in dietary pattern. They concluded that urban life style and diets may predispose to a higher incidence of diabetes, hypertension and atherosclerotic CVD among tribal migrants living in urban areas.[59]

In conclusion after having studied many publications about the nutrition transition it has become clear that not only in SA but also in many developing countries certain changes take place in dietary areas when people migrate from rural to urban areas. These mainly include a greater consumption of animal protein, fat, and sugar while their carbohydrate and fibre intake decreases.

2.1.2 PHYSICAL ACTIVITY

Physical activity reduces CHD risk by retarding athrogenesis, increasing the vascularity of the myocardium, increasing fibrinolysis, and modifying other risk factors such as increasing HDL-C, improving glucose tolerance and insulin sensitivity, aiding in weight management, and reducing BP. A sedentary person has twice the risk of developing CHD as a person who is active.[17] Globally, physical inactivity is recognized as the fourth leading risk factor of mortality and is responsible for 6% of NCD risk such as CHD, type 2 diabetes, breast cancer and colon cancer.

According to estimates, six out of ten deaths, currently, are attributable to physical inactivity and NCD are responsible for nearly half of the overall global burden of disease.[60] Physical inactivity, while recognized globally as a major risk factor in the morbidity and mortality resulting from NCD, would appear to be insufficiently appreciated in developing and developed countries. Research data on the magnitude and impact of physical inactivity and cardiovascular fitness in SSA, including SA remains sparse.[61]

In the THUSA study, Oosthuizen et al. (2002)[62] examined the impact of urbanisation on serum lipid profiles. They found that serum lipid levels increased with urbanisation in males and females, although they remained within levels recommended for other populations. The main factor responsible for these increases was an increased BMI which was probably associated with decreased PA. The relationship between PA and the prevalence of risk factors for CVD in communities undergoing rural to urban transition was also assessed as part of the THUSA study.[63] Their main findings were an inverse association between PA and CVD risk factors, especially among females, and they recommended that deterrents to PA in black females should be identified, as PA was seen to provide some protection against CVD, even in overweight subjects.

In the 2012 SANHANES survey, researchers found that almost two-thirds of male participants (62.4%) were physically fit, compared to 42% of female participants.[8] More than 57% of male participants in each age group, 18–24 years (65.9%), 25–29 years (61.9%;), and 30–40 years (57.1%) were found to be physically fit. However, a notable but statistically insignificant reversal of this trend was found among female participants where 38% of those in the age group, 18–24 years, who tested being fit, increased to 45.8% in the 25–29-year age group and 45.0% in the 30–40-year age group. Thus, the percentage of physically fit females increased with age, which was the opposite to what was found in the males. Therefore, in summary, one out of four males (27.9%) and one out of two females (45.2%) were unfit.[8]

Muhihi et al. (2012)[64] investigated the PA level and its relationship with CVD risk factors among young and middle-aged males in a fast growing city of Mwanza, Tanzania. Physical activity was assessed among 97 healthy males, age 20–50 years, using the SSA Activity Questionnaire. An updated compendium of PA was used to code the metabolic equivalents. Energy expenditure was calculated using the Harris Benedict equation. Anthropometric measurements, BP, fasting blood glucose and serum lipids were also measured.

They found that the mean energy expenditure in this population was 6466 ± 252 kcal/week(27 157 ± 1058kJ). More than half (53.6%) of the participants had energy expenditure of more than 4000 kcal/week(16 800kJ). Only three (3.1%) had an energy expenditure below the recommended 1000 kcal/week. The prevalence of the various CVD risk factors was as follows, hypertension 23.7%, low HDL-C 10.3%, high LDL-C 9.3%, and obesity 4.1%.

Physical activity energy expenditure had an inverse relationship with the waist-hip-ratio (WHR), systolic BP (SBP) heart rate, TC, HDL-C, LDL-C, TG and fasting blood glucose. They, thus, concluded that PA energy expenditure was high and inversely correlated with CVD risk factors and may play an important role in the prevention of CVD in this urban population of young and middle-aged males.

Peer et al. (2013)[65] described the urban-rural and gender patterns of NCD risk factors in black adolescents and young adults (15–24-year-olds) from two South African Demographic and Health Surveys (SADHS) conducted five years apart. They found that urban youth, compared to their rural counterparts, were more likely be physically inactive (OR: 1.45, 95% CI 1.12-1.89). Low levels of PA, obtained in 2003 but not in 1998, were higher in females (46.6%) compared to males (31.3%). When evaluating the risk factors in 2003, physical inactivity was the predominant contributor to NCD risk, ranging from 27.1% in rural males to 53.4% in urban females.

The PA patterns of a cohort of middle-aged urban-dwelling black African females were assessed to determine whether PA was associated with anthropometric measures and metabolic outcomes.[66] Gradidge et al. (2014)[66] made use of a cross-sectional sample of 977 black African females (mean age 41.0 ± 7.84 years) from the Bt20 study based in Soweto, Johannesburg. They found that the prevalence of metabolic syndrome in this sample was 40.0%, and the prevalence of overweight and obesity was 29.2% and 48.0%, respectively. Females who reported owning a motor vehicle, “walked” less and participated in more leisure-time activity (both p <0.01), while females, who owned a television reported significantly lower moderate-vigorous PA and walking (both p <0.01). Sitting time (mins/wk) was not different between the activity groups, but was associated with TG and diastolic BP (DBP). Total PA was inversely associated with fasting insulin, and PA in the work domain was associated with fat free soft tissue mass.[66]

They concluded that most urban dwelling black South African females are classified as physically active despite a high prevalence of obesity and metabolic disease risk factors. Furthermore, sitting time had detrimental effects on TG levels and DBP, while total PA attenuated fasting insulin levels. Gradidge et al. (2014)[66] recommended that since walking is a major contributor to PA, future research should attempt to determine whether the intensity of this activity plays a role in the prevention of cardio-metabolic diseases.

Afrifa-Anene et al. (2015)[67] assessed BP levels and examined its association with PA and BMI among urban poor youth in Accra, Ghana. Participants (n=201), age 15–24 years, came from three poor urban communities. Their height, weight and BP were measured, while PA levels were assessed using the Edulink Urban Health and Poverty project questionnaire. Multiple linear regression analysis was used to determine the factors influencing BP levels. They found that the proportion of pre-hypertension and hypertension among the youth was 32.3% and 4%, respectively. The rates of pre-hypertension (42.0 vs. 24.8) and hypertension (6.8 vs. 1.8) were higher in males than in females and more than three-quarters (84.1%) were not physically active. Females were found to be more physically inactive compared to the males (94.7% vs. 70.5%). Females average BMI was 22.8 kg/m^2, and female rates for overweight (17.7 vs. 6.8) and obesity (13.3 vs. 2.3) were higher than those for males. Body mass index was significantly associated with SBP (β=1.4, p <0.000; and β=0.8, p <0.000, respectively for male and female youth) compared to DBP. Youth with low PA had a raised BP. They, ~~thus,~~ concluded that the positive association of BMI and BP in their study participants suggests the need for further studies on BP and other risk factors. This is necessary among the youth of rural populations and other developing countries to stall the rising prevalence and implications for adult morbidity and mortality.[67]

2.1.3 TOBACCO USE

Cigarette smoking is a major arteriosclerotic risk factor, which is enhanced by the presence of diabetes mellitus.[68] Therefore smoking is a risk factor that is related to high rates of CHD mortality. Key facts from the WHO Tobacco Fact Sheet (2016) indicate that “Tobacco kills up to half of its users and kills around 6 million people each year”.[69] More than 5 million of those deaths are the result of direct tobacco use, while more than 600 000 are the result of non-smokers being exposed to second-hand smoke. Also, nearly 80% of the world's 1 billion smokers live in LMIC, where the burden of tobacco-related illness and death is heaviest. Furthermore, tobacco users who die prematurely deprive their families of income, raise the cost of health care and hinder economic development.

Globally, tobacco smoking causes about 71% of lung cancer, 42% of chronic respiratory disease and nearly 10% of CVD.[70] The US Centers for Disease Control (CDC) maintains that compared to non-smokers, smoking increases the risk of CHD by 2 to 4 times, stroke by 2 to 4 times, lung cancer for males by 23 times and for females by 13 times, and dying from chronic obstructive lung diseases (such as chronic bronchitis and emphysema) by 12 to 13 times.[71] While the prevalence of smoking is considerably higher in males than in females, second-hand smoke disproportionately harms females.

In the SANHANES survey, the smoking behaviour of the South African population was studied.[8] Overall, 20.8% of the population had a reported history of having smoked tobacco and 79.2% had never smoked tobacco. Smokers comprised those who are daily smokers (16.2%), ex-smokers (2.6%), and occasional smokers (2.0%). The mean age of initiation of smoking tobacco for males was significantly lower than that of females (16.4 years compared to 17.9 years of age). The mean age of initiation of using other tobacco products was lower for males than females (21.5 compared to 27.9 years of age). The mean number of other tobacco products used per day was significantly higher for males than females (20.3 compared to 7.7). Males reported an ever smoking tobacco rate of 32.8%, which is three times more than that of females at 10.1%. The prevalence of having ever smoked tobacco increased with age up to the 55–64-years age group, then decreased after age 65 years and older. Individuals from rural formal localities reported the highest rate of having ever smoked tobacco (24.5%), however, this was not significantly different from the national rate (20.8%).

Western Cape residents reported the highest provincial rate of having ever smoked tobacco (38.5%), which was significantly higher than the national prevalence (20.8%). Limpopo residents reported the lowest rates of having ever smoked (14.4%), which was significantly lower than the national average (20.8%). Coloured individuals reported the highest rate of having ever smoked (44.9%), which was significantly higher than the national average (20.8%).

Peer et al. (2013)[65] described the urban-rural and gender patterns of NCD risk factors in black adolescents and young adults (15- to 24-year-olds) from two South African Demographic and Health Surveys conducted 5 years apart.[65] Changes in tobacco use among 15–24-year-olds as well as urban-rural and gender differences were analysed in the SADHS conducted 5 years apart.[65] In males, the prevalence of smoking (1998: 21.6%, 2003: 19.1%) was high and increased with age, but in females this was much lower (1998:1.0%, 2003: 2.1%;).[65]

In the present study, Peer et al. (2014)[72] reported that the age-standardized prevalence of smoking ≥1 cigarette/day was 48.5% (95% CI 43.0–54.0) in males and 7.8% (95% CI 5.8–10.5) in females (p <0.001). The prevalence in males was found to be lower in 2008/09 (51.0%, 95% CI 45.2–56.7) compared to 1990 (59.7%, 95% CI 53.8–65.4) but unchanged in females (2008/09: 8.0%, 95% CI 5.9–10.7; 1990: 8.4%, 95% CI 6.0–11.8).

In the logistic model for males, smoking was associated with younger age (p=0.005) and being poor (p=0.024). In females, spending more than half their lives in the city (p <0.001), being poor (p=0.002), and coping poorly with stress [defined by lower SOC (Antonovsky's sense of coherence score)]; *OR*: 1.04, 95% CI 1.01–1.08; p=0.035) were associated with smoking. The increasing number of adverse events, which replaced SOC in the same models, was significant for females (*OR*: 1.10, 95% CI 1.01–1.21; p=0.047) but not for males. They concluded that the high smoking prevalence in males and unchanged rate in females require additional interventions to curtail this behaviour.[72]

2.2 INTERMEDIATE RISK FACTORS

2.2.1 RAISED BLOOD PRESSURE (HYPERTENSION)

Hypertension is a common condition in SA and is a risk factor for heart attacks and stroke.[74] In the present study hypertension is defined as a SBP equal to or above 140 mmHg and/or a DBP equal to or above 90 mmHg.[17]

A high salt (sodium chloride) intake and obesity are known to contribute to the development of raised BP and hypertension. Seedat et al. (1996)[18] suggested that black African adults have an abnormal transport mechanism of Na and rennin activity, thereby increasing their risk for hypertension. A high Na intake is common in SA, particularly in poor settings, as it is used to preserve food or to make food tastier.[17] Food items high in Na such as flavour enhancers (e.g. Aromat and soup powders) are added to many dishes as they create a meaty taste, thereby considerably increasing Na intake.[17] On the other hand, potassium and calcium have a protective effect and high levels are encouraged in the diet.[74]

Charlton et al. (2005)[74] investigated whether habitual intakes of Na, potassium, magnesium, and calcium differ across South African ethnic groups. This was a cross-sectional study on 325 black, white, and Coloured hypertensive and normotensive subjects. Mean urinary Na excretion values equated to daily salt intakes of 7.8 g, 8.5 g, and 9.5 g in black, coloured, and white subjects, respectively (p <0.05). Between 33% and 46% of total Na intake was discretionary (added at the table), and of the non-discretionary sources, bread was the single greatest contributor to Na intake in all groups. Ethnic differences in calcium intake were evident, with black subjects having particularly low intakes. Urban versus rural differences regarding sources of dietary Na existed, with greater than 70% of total non-discretionary Na being provided by bread and cereals in rural black South Africans compared to 49% to 54% in urban dwellers. Charlton et al.(2005)[74] concluded that white South Africans have higher habitual intakes of Na, but also higher calcium intakes than their black and Coloured counterparts. All ethnic groups had Na intakes in excess of 6 g/d of salt, whereas potassium intakes in all groups were below the recommended level of 90 mM/d. They also concluded that dietary differences may contribute to ethnic-related differences in BP.

A dietary approach is an effective way to both prevent and treat high BP, as there is strong evidence of an association between Na intake and high BP.[75,76] High intake of salt is also strongly linked to the risk of IHD and stroke.[75] The mean salt intake is estimated to be about 8–10 g per day in Western countries, but this figure may be much higher in many Eastern European and Asian countries.[77] The WHO recommends a salt intake of less than 5 g (about one teaspoon) per day, which yields <2000 mg Na per day.[78] Pooled data from a meta-analysis estimated a significant fall in SBP and DBP of 5.4/2.8 mmHg in hypertensive patients, and 2.4/1.0 mmHg in normotensive patients, respectively, with a modest reduction in salt intake over 4 weeks or longer.[79]

A major reduction in salt intake requires a major decrease in the salt content of processed foods. Charlton et al. (2005)[74] found that in addition to bread being the highest contributor of salt, meat products such as processed meats (polony, a pork-based product; Vienna sausages; salami; ham; and other sausages), commercial meat pies, and margarine (brick type), were also important sources. A stepwise approach in reducing the salt content of processed food could be the key to achieving recommendation goals without consumer resistance. A 5% reduction per week in the Na content of bread, for 6 weeks, can be achieved without losing consumer acceptance and has been found to result in a one-quarter reduction in the Na content of bread, a major source of Na in the diet.[74]

The Dietary Approaches to Stop Hypertension (DASH) diet was designed to lower BP.[80] The DASH diet constitutes a high intake of fruit, vegetables, low-fat dairy, wholegrain cereals, legumes, fish, and poultry with a reduction in salt, sugar and fat and increased levels of potassium and calcium. Research has indicated that this diet regimen decreases BP.[81,82] The DASH diet can be combined with a lowered salt intake for even greater effectiveness. Several other dietary and lifestyle factors also play an important role in hypertension. Obesity is the most important one, followed by lack of PA as well as an excessive alcohol intake.

Relatively low hypertension rates compared to other urbanised black studies in SA, i.e. 9.2% in males and 12.9% in females, were reported in the BRISK study.[83] Bourne et al. (1993)[34] indicated that 47% of their respondents liked their food well-salted and salty snacks were consumed more than three times per week by 36% of individuals.

In the SANHANES study, the mean SBP for males (130.1 mmHg) was found to be significantly higher than for females (127.6 mmHg), but DBP did not differ significantly by sex.[8] The mean SBP increased progressively with increasing age from 118.1 mmHg for the 15–24-year age group to over 149.3 mmHg in the 65-years and older age group. Across localities, urban informal (124.0 mmHg) and rural informal communities (127.9 mmHg) had the lowest mean SBP, while across provinces, the mean highest SBP was recorded in the Western Cape (131.8 mmHg), Free State (133.9 mmHg) and

North West (131.0 mmHg). The white (130.8 mmHg) and coloured (132.1 mmHg) groups had the highest mean SBP. Overall, a similar pattern was seen for the mean DBP. Furthermore, the rural formal areas consistently had the highest pre-hypertensive and hypertensive SBP and DBP when compared to other areas of residence, overall. The provincial rates of systolic pre-hypertension ranged between 32.4% (KwaZulu-Natal) and 46.1% (Free State), while the provincial diastolic pre-hypertensive rates ranged from 14.5% (Limpopo) to 27.3% (Western Cape). The systolic hypertensive rates ranged from 19.0% to 29.4% and diastolic hypertensive BPs ranged from 8.3% to 19.4% across the provinces. By race, the black African and Indian race groups had the lowest rates of pre-hypertensive and hypertensive BP.

In the present study, Peer et al. (2013)[84] reported that the age-standardised hypertension prevalence was 38.9% (95% CI 35.6–42.3) with similar rates in males and females. Among the 25–64-year-olds, the hypertension prevalence was significantly higher in 2009 (35.6%, 95% CI 32.3–39.0) than in the 1990 BRISK study (21.6%, 95% CI 18.6–24.9). They found that in 2009, hypertension odds increase with age, family history of hypertension, higher BMI, problematic alcohol intake, physical inactivity and urbanisation. Also, among hypertensive participants, significantly more females than males were detected (69.5% vs. 32.7%), treated (55.7% vs. 21.9%) and controlled (32.4% vs.10.4%) in 2009. There were minimal changes from 1990 except for improved control in 25–64-year-old females (1990:14.1% vs. 2009:31.5%).

2.2.2 RAISED BLOOD GLUCOSE (DIABETES)

Diabetes is a disease condition that occurs when the pancreas does not produce enough insulin, or when the body cannot effectively use the insulin it produces.[85] This leads to an increased concentration of glucose in the blood or hyperglycaemia which can result in diabetes. Type 1 diabetes is characterised by a lack of insulin production. Type 2 diabetes is caused by the body's ineffective use of insulin, which often results from excess body weight and physical inactivity.

Diabetes is routinely diagnosed by testing an individual's blood for glucose (sugar) after an overnight fast or after performing a glucose tolerance test under the same conditions. Glycated haemoglobin is the current gold standard test to monitor long-term blood sugar control in people already diagnosed with diabetes. Recently, HbA1c has been proposed by the WHO as another test to detect diabetes in undiagnosed people.[86] The WHO recommends that "an HbA1c of 6.5% is the cut-off point for diagnosing diabetes". A value of less than 6.5% does not exclude diabetes and needs to be diagnosed using glucose tests. With regard to the latter part of the recommendation, the use of a lower cut-off of 6.1% has been recommended as being appropriate in the screening and diagnosis of diabetes.[8]

It is well-established that there is an association between a high kilojoule diet (energy-dense) and increased BMI, which is associated with the development of type 2 diabetes. Despite this, until recently there has been less evidence regarding an association with other dietary factors. However, recent studies have shown a significant association between the development of type 2 diabetes and sugar-sweetened beverages.[87] In 2012, three databases (PubMed, Cochrane Library, and Science Direct), and the SA Journal of Clinical Nutrition (SAJCN), DOH and SA Medical Research Council (SAMRC) websites were searched for SA studies on sugar intake published between January 2000 and January 2012.[87] Studies were included in the review if they evaluated sugar intake and dental caries; sugar intake and non-communicable diseases; sugar and diabetes; sugar and obesity and/or sugar and micronutrient dilution. Steyn et al. (2012) found that the intake of sugar appeared to be increasing steadily across the South African population. Data from numerous systematic reviews showed that dietary sugar increases the risk for development of obesity and type 2 diabetes and that the risk for development of these conditions appeared to be especially strong when sugar was consumed as sugar-sweetened beverages.[87]

Malik et al. (2013)[88] conducted a systematic review and meta-analysis to summarize the evidence. Thirty-two original articles were included in the meta-analyses; 20 in children (15 cohort studies, n=25 745; 5 trials, n=2772) and 12 in adults (7 cohort studies, n=174 252; 5 trials, n=292). In cohort studies, one daily serving increment of SSBs (sugar sweetened beverages) was associated with a 0.06 (95% CI 0.02, 0.10) and 0.05 (95% CI 0.03, 0.07) unit increase in BMI in children and 0.22 kg (95% CI 0.09, 0.34 kg) and 0.12 kg (95% CI 0.10, 0.14 kg) weight gain in adults over 1 y in random- and fixed effects models, respectively. RCTs (random controlled trials) in children showed reductions in BMI gain when SSBs were reduced [random and fixed effects: 20.17 (95% CI 20.39, 0.05) and 20.12 (95% CI 20.22, 20.2)], whereas RCTs in adults showed increases in body weight when SSBs were added (random and fixed effects: 0.85 kg; 95% CI 0.50, 1.20 kg). They conclude that their review provided evidence that SSB consumption promotes weight gain in children and adults.

Diet remains the cornerstone of the management of type 2 diabetes. Intensive therapy, consisting of a combination of medication, diet, and PA, effectively delays the onset of DM (diabetes mellitus), manages existing DM, and slows the development and progression of complications in patients with insulin-dependent DM.[89]

2.2.3 ABNORMAL LIPID VALUES

Cholesterol is required by the body in the synthesis of hormones and also for bile production, which is necessary for the digestion of fats.[17] The dietary intake of cholesterol varies with the food choices of an individual and is, therefore, a modifiable risk factor in the development of CVD. There are two types of cholesterol – HDL-C, the so-called good cholesterol, and LDL-C, the so-called bad cholesterol. Low-density lipoprotein cholesterol is responsible for the development of CVD, whereas HDL-C is thought to provide protection against this disease process. Therefore, it is important to track this dietary risk factor to measure changes in the blood levels of cholesterol over time and design appropriate interventions.

Research indicates that weight loss induced by a weight-reducing diet and regular exercise reduces TC, LDL-C, and BP, and increases HDL-C.[90,91] Total lipids (total fat) consists of different types of fatty acids, such as SFA, MUFA, and PUFA, as well as other components, such as cholesterol. The type of fat consumed is of far more importance in the development of IHD than the total amount of fat consumed. This is because the different types of fatty acids affect the blood lipids and lipoproteins differently.[92] A total fat intake of at least 20% of total energy (20% E) is regarded as consistent with good health, and women of reproductive age need at least 20% E from fat.[78] In the United States, a total fat intake of 25% to 35% E is recommended as part of the Therapeutic Lifestyle Changes (TLC) dietary goals recommended by the National Cholesterol Education Program (NCEP) of the Adult Treatment Panel (ATP) III for the prevention of CVD.[94]

A high consumption of SFA raises total cholesterol and LDL-C, although different types of SFA have different effects.[93] The major sources of SFA are dairy and meat foods which have a high fat content. Other important sources of SFA are tropical oils, viz. palm kernel oil and coconut oil. Tropical oils, therefore, are best avoided. In many countries the presence of these vegetable oils is stated on food labels. However, removing these oils from the diet can be problematic as they are an important source of low-cost food energy for poor people in many countries.[78]

An intake of SFA of <10%E is recommended by the WHO for the prevention of IHD.[78] The TLC dietary goals, which are directed at people with elevated LDL-C concentrations, recommend that SFA intake should be reduced to <7%.[98] Polyunsaturated fatty acids are partially hydrogenated in order to increase the shelf-life of foods containing these fatty acids and improve the texture. This process leads to the formation of trans-fatty acids. These fats are mostly found in retail fats (such as hard margarine), spreads, deep-fried fast foods, and baked goods. Naturally occurring trans-fatty acids are found in small amounts in dairy products and in meat from ruminants. Research indicates that trans-fatty acids increase the risk of IHD because they elevate LDL-C and decrease HDL-C.[94] Trans-fatty acids are now recognized as the most dangerous type of dietary fat in terms of increasing the risk of IHD. The intake of trans-fatty acids should therefore be minimized, and should be under 1% TE.[93,95] MUFAs contain one double bond in the carbon chain. Many foods contain MUFAs, but olive oil is an especially rich source of oleic acid, a MUFA. Polyunsaturated fatty acids contain two or more double bonds in the carbon chain. There are two families of PUFA in the diet, the omega-6 (n-6), of which linoleic acid (LA) is the parent fatty acid, and the omega-3 PUFA (n-3), of which α-linolenic acid (ALA) is the parent fatty acid. Both omega-6 and omega-3 fatty acids are referred to as essential fatty acids because they are required for various body functions but cannot be formed in the body and must, therefore, be consumed as part of the diet.

Over the past decades, large-scale studies repeatedly have demonstrated a definite association between types of dietary fat and elevated blood lipid levels, especially cholesterol.[78] Raised TC and LDL-C and/or low HDL-C are important risk factors for developing IHD.[78,96] While excessive intake of dietary cholesterol can contribute to a raised blood cholesterol level, it has been shown that SF have a far greater impact on TC and LDL-C levels.[78] High blood cholesterol levels are caused primarily by eating too much SF and eating too little soluble fibre.[78] Elevated TG levels are also an independent risk factor for CHD. High blood TG levels are caused primarily by one or more factors, i.e. eating too much fat, eating too many refined CHO foods, and drinking too much alcohol.

The risk-factor profile for NCD in the older (age >60 years) black African population of Cape Town was found to be low in the BRISK study in 1999.[101] They had low TC and LDL-C levels as well as high levels of HDL-C. Only 7.3% of the males and 11.3% of the females had moderately raised LDL-C (3.4–5.2 mmol/l) and none had high levels of LDL-C (>5.2 mmol/l). This, in turn, resulted in 90% of the sample having protective levels of HDL-C/TC ratios.[97] However, this was not comparable to the younger age groups.

Oelofse et al. (1996)[98] found that 25% of the females had TC levels above the cut-off level for moderate CHD risk. Sixteen per cent of both the males and females had HDL-C levels of <1 mmol/l. They also found that the females had higher TC levels than males in each age category (15–24, 25–34, 35–44, 45–54, 55–64, 15–64), which could be explained by the high prevalence of obesity among females in the study population. Based on the increased urbanisation of this population, it is possible that the lipid profile of the Cape Town blacks may have changed considerably, despite the fact that they have been protected by their past high HDL-C levels.

The recent SANHANES study examined dyslipidaemia in all population groups.[8] Of the black African males, 15% had abnormal serum cholesterol levels (>5 mmol/L), 53.3% had abnormal HDL-C levels (<1.2 mmol/L), 15% had high LDL-C levels (>3 mmol/L), and 25.1% had high TG levels (>1.7 mmol/L). In female participants, the prevalence of abnormal lipid concentrations was even higher. Of the black African females, 24.9% had abnormal serum cholesterol levels (>5 mmol/L), 26.4 had high LDL-C levels (>3 mmol/L), and 45.4% had abnormal HDL-C levels (<1.2 mmol/L). The pattern of abnormally elevated TG was similar in extent to that of other lipids.[8]

Peer et al. (2014)[99] determined the prevalence, determinants, and management of dyslipidaemia in the present study of the urban black population of Cape Town and examined the changes between 1990 and 2009 in the 25–64-year-old sample. The prevalence of raised TC, raised LDL-C, and reduced (HDL-C) were 25.2%, 37.8% and 55.2% in males and 23.1%, 47.0% and 66.8% in females, respectively. Between 1990 and 2009, the raised LDL-C and reduced HDL-C prevalence increased significantly with no change for raised TC. Among participants with raised LDL-C, only 2.6% were aware of their diagnosis, 2.7% were on treatment, and 1.5% had LDL-C <3 mmol/l. In the logistic model, increasing age (OR 1.04, 95% CI 1.03–1.05; p <0.001), rising BMI (OR 1.03,95% CI 1.01–1.05; p <0.003), and fat intake more than 30% of diet (OR 1.37, 95% CI 1.02–1.85; p <0.035) were/was significantly associated with LDL-C >3 mmol/l, but not sex, PA, or urbanisation. The researchers concluded that the dyslipidaemia pattern in this population requires full lipogram screening in high-risk individuals and demands improved management using a total CVD risk approach.

2.2.4 OVERWEIGHT/OBESITY

The epidemic of obesity can be explained in terms of energy balance to some degree. Therefore, the real question is, 'Why is much of the world's population eating more food energy than they burn up'? Decades of research have given us a reasonably good answer to this question. A major factor leading to obesity is lack of or low levels of PA. This may occur as a result of the combination of urbanisation and labour-saving machinery.[100]

Over recent decades, many tens of millions of people have relocated to the cities. Most jobs today require far less expenditure of energy. At the same time, thanks to the availability of cars and buses, people today typically walk much less than they used to. Another major cause of obesity is the widespread availability of highly palatable, energy-dense food (i.e., high quantity of kJ per gram). A large amount of accumulated evidence demonstrates how such food leads to excess intake of food energy, in other words, over nutrition. Such foods have four key features, e.g. a high fat content, a high refined sugar content, a low fibre content, and a high energy density. These features of the modern, Western diet should not be viewed singly: they act synergistically.[100]

Most human studies indicate that a high-fat diet induces excessive energy intake and, hence, weight gain.[101] This has been repeatedly shown in epidemiological and experimental investigations. One study that illustrates this, was the Women's Health Initiative Dietary Modification Trial, for which 49 000 post-menopausal women were recruited. The study's aim was to investigate whether a low-fat diet reduces the risk of cancer. Accordingly, half the women were instructed to lower their intake of fat, but were not advised to lose weight or to take exercise. The investigators discovered that when women lowered their fat intake by 11% (e.g., from 40% of energy to 29%), their weight decreased over the following year by about 1.4 kg.[102]

The next villain in the obesity epidemic is sugar. In particular, sugar-sweetened beverages have a similar effect on energy balance as does dietary fat; consuming these drinks leads to spontaneous overconsumption of food energy.[88,103] Several obesity experts have suggested that a major cause of the obesity epidemic among American children and teenagers over the past 30 years is the greatly increased consumption of soft drinks.[88] With respect to weight control, fruit juices, as far as is known, have no advantage over soft drinks.

Fibre has an opposite action in the body as sugar and fat; the presence of fibre in foods tends to induce satiation (a feeling of fullness), thereby bringing about a halt to eating. The role of fibre in retarding the development of obesity is supported by strong epidemiologic evidence. Numerous studies have reported that the intake of dietary fibre is inversely associated with body weight and body fat.[104] An important factor that determines the satiating ability of a particular food is its energy density.[105] Foods with more concentrated energy (more kJ per gram) have less satiating power (i.e., little appetite satisfaction relative to energy consumed), and they are, therefore, more likely to lead to overconsumption of food energy. This may be a major reason why fat causes weight gain: because fat has more than twice as much energy per gram as either protein or carbohydrates, fat-rich foods tend to be energy dense. Conversely, foods with a high water content have a low energy density and can, therefore, satiate the appetite before much food energy has been consumed. Many types of fruit and vegetables, such as apples, melon, carrots, and cabbage, have a high water content and are, therefore, particularly good at satisfying the appetite.

In summary, doughnuts and biscuits (cookies) are a mixture of fat, sugar, and refined flour, with a minimal content of water and fibre. These are the type of foods that readily cause people to overeat and become overweight. Another factor deserving mention in the discussion of the obesity causes, is portion sizes. These have been steadily expanding for the past 40 years. For example, plate sizes in restaurants are significantly larger now than they were a few decades ago. In the case of bottle sizes for cola drinks, these are now three or four times larger. These observations are based mainly on studies in the United States. But trends that start there frequently spread to many other countries. This is potentially important because evidence shows that when people have more food placed in front of them, they eat more. This problem appears to be additive when combined with increased energy density.[106]

Popkin et al. (2012)[107] reported that decades ago discussion of an impending global pandemic of obesity was thought of as heresy. Diets in the 1970s began to shift towards increased reliance upon processed foods, increased away from home intake and greater use of edible oils and sugar-sweetened beverages. Reduced PA and increased sedentary time was seen too. These changes began in the early 1990s in the LMIC but did not become clearly recognized until diabetes, hypertension and obesity began to dominate the globe. Urban and rural areas from SSA and South Asia’s poorest countries to the higher income ones are shown to have experienced rapid increases in overweight and obesity status. Concurrent rapid shifts in diet and activity are documented. An array of large-scale program and policy shifts are being explored in a few countries; however, despite the major health challenges faced, few countries are serious in addressing prevention of the dietary challenges faced.

Overwhelming evidence continues to accumulate that obesity is associated with significant morbidity and mortality and in particular that it is an independent risk factor for CVD.[108] The association of obesity with CVD and its risk factors, including hypertension, dyslipidaemia, and glucose intolerance is becoming more clearly understood and an increasing body of evidence indicates that risk factors tend to cluster in obese individuals and may act synergistically to increase their risk for CVD.[108]

The Framingham Heart Study was initiated in 1948 to study the factors associated with the development of CVD by employing long-term surveillance of an adult population in Framingham, Massachusetts.[109] This was designed as a longitudinal investigation of constitutional and environmental factors influencing the development of CVD in males and females free of these conditions at the outset. Changing patterns of cigarette smoking, nutritional habits and exercise patterns as well as changes in other aspects of lifestyle and advances in medical care may all influence future morbidity and mortality rates for CVD. The Framingham Offspring Study was initiated to assess familial and genetic factors as determinants of CHD. Participants were drawn from the original and offspring cohorts of the study to assess the lifetime risk of CVD among individuals with and without obesity and diabetes.[110] Over 30 years, the lifetime CVD among females with diabetes was 54.8% among normal-weight females and 78.8% among obese females. Among normal-weight males with diabetes, the lifetime risk of CVD was 78.6%, whereas it was 86.9% among obese males. They have concluded that the lifetime risk for CVD was the highest among obese participants who were also type 2 diabetics. Obesity has been shown to be an independent risk factor for cardiovascular morbidity and total mortality.[110]

The association between body weight and both overall morbidity and mortality from specific causes was examined in a cohort of 115 195 US middle-aged females enrolled in the prospective Nurses' Health Study in the USA.[111] The researchers concluded that body weight and mortality from all causes were directly related among these females. They also found that lean females did not have excess mortality. The lowest mortality rate was observed among females who weighed at least 15% less that the US average for females of similar age; and among those whose weight had been stable since early adulthood.

The prevalence of type 2 diabetes in the United States is significantly higher in a number of ethnic groups compared to Caucasians, particularly among African-American women, who have twice the diabetes prevalence of Caucasians in some age groups. Lovejoy et al. (1996)[112] compared the insulin sensitivity of African-American and Caucasian females, matched for age, degree of obesity and WHR. They further investigated intra-abdominal fat distribution and health risk factors between the two populations. They found the visceral fat area was smaller in African-American females compared to those in Caucasians. Also, visceral fat area strongly correlated with WHR in the Caucasian females but not in the African-Americans. Furthermore, visceral fat correlated with metabolic risk factors in both groups, but subcutaneous abdominal fat significantly correlated with the insulin sensitivity index and fasting insulin only in the African-American females. Their results suggested that the relationship between body fat distribution and health risk factors may be different in African-Americans and Caucasians and that reduced insulin sensitivity in African-American females may, in part, explain the high diabetes rate in this population.

Adeboye et al. (2012)[113] examined the evidence of obesity and its association with co-morbidities within the African continent. Comparative studies conducted in Africa on adults 17 years and older with mean BMI ≥28 kg/m^2 were included. Five electronic databases were searched. Surveys, case–control and cohort studies from January 2000 to July 2010 were evaluated. Of 720 potentially relevant articles, 10 met the inclusion criteria. Prevalence of obesity was higher in urban than rural subjects with significant increases in obesity rates among females. Inflammatory marker levels were significantly elevated among Africans compared to Caucasians. The co-relationship between obesity and chronic diseases was also highlighted. They demonstrated that while obesity remains an area of significant public health importance to Africans, particularly in urban areas, there is little evidence of proper diagnosis, treatment and/or prevention.[113]

In the BRISK study of 1990 in Cape Town 7.9% of the men and 34.4% of the females were obese.[33] Similarly, in the SADHS of 2003, 9% of South African males and 27% of South African females were found to be obese, while 8% of black African urban males and 34% of black African females were obese, compared to 5% of black African rural males and 21% of black African rural females.[114] The most recent national data from the SANHANES indicated that 18.7% of black African males were overweight and 10.1% were obese and 25% of black African females were overweight and 40.7% were obese.[8] In African females, 43.2% of urban females and 37.2% or rural females were obese. In males this was 14.5% vs. 10%.

Micklesfield et al. (2013)[115] reported that SA is undergoing a rapid epidemiological transition and has the highest prevalence of obesity in SSA, with black females being the most affected (obesity prevalence 31.8%). Although genetic factors are important, socio-cultural, environmental and behavioural factors as well as the influence of SES more likely explain the high prevalence of obesity in black South African females. Their review examined these determinants in black South African females, and compared them to their white counterparts, black South African males, and where appropriate, to females from SSA. This review specifically focused on environmental factors influencing obesity, the influence of urbanisation as well as the interaction with socio-cultural and socio-economic factors. In addition, the role of maternal and early life factors and cultural aspects relating to body image were also discussed and ~~thus~~ this information can be used to guide public health interventions aimed at reducing obesity in black South African females.

The authors concluded that there is compelling evidence that the prevalence of obesity is increasing in SSA, and that this increase is linked to urbanisation, economic development and concomitant lifestyle risk factors, such as physical inactivity and poor dietary practices. In addition, there are a number of paradoxes that have emerged, including the positive association between food insecurity and obesity, the non-linear association between education and obesity, as well as the distinct differences between patterns and determinants of obesity in males and females in the region. Steyn and Mchiza's[116] review on obesity and the nutritional transition in SSA, illustrates the outcomes of this transition and its association with overweight and obesity, along with exploring the relationship with the double burden of malnutrition.[116] They described the increase in overweight in nearly all SSA countries and presented data on associated increased GDP, and availability of energy, protein, fat, and sugar at country national levels. However due to poverty not everyone may have access to this food. Predictors of overweight are described by means of various studies undertaken in SSA, and dietary intakes of numerous countries are presented. Overall, they showed that SES, gender, age, parity, physical inactivity, and increased energy, fat, and sugar intake are powerful predictors of overweight and/or obesity. The urgency for health interventions in countries in the early stages of the nutrition transition is emphasized, particularly in view of the fact that fat intake is still less than 30% E intake in nearly all sub-Saharan countries.

In the SANHANES study, SA males were found to have a mean BMI of 23.6 kg/m^2, which was significantly lower than that of females (28.9 kg/m^2).[8] The prevalence of overweight and obesity was significantly higher in females than males (24.8% and 39.2% compared to 20.1% and 10.6%, respectively). On the other hand, the prevalence of underweight and normal weight was significantly higher in males than females (12.8% and 56.4% compared to 4.2% and 31.7%, respectively). The highest mean BMI was seen in females in the 45–54 and 55–64-year-old age groups. In females, the highest prevalence of overweight (28.0%) and obesity (56.3%) was seen in the 25–34 and 45–54-year-old age groups, respectively. The prevalence of obesity in females, 45–54 years old, was significantly higher than in females in the younger age groups (56.3% compared to 8.0%, 21.7%, 36.3%, and 44.8%). Males living in urban formal areas had a significantly higher mean BMI (24.3 kg/m^2) compared to those in the other areas, and were 2.1 and 1.5 times more likely to be obese than those living in urban informal and rural informal areas (13.2% compared to 6.3% and 8.7%, respectively). Females living in urban formal areas had the highest mean BMI (29.4 kg/m^2) as well as the highest prevalence of obesity (42.2%). The prevalence of overweight was highest in females living in an urban informal area (27.9%). Rural formal areas had the lowest prevalence of obesity (31.8%). They also found that one in ten males (9.8%) had a waist circumference equal to or larger than 102 cm, while 50.8% of females had a waist circumference equal to or larger than 88 cm.

Sartorius et al. (2015)[117] identified contextualised high impact factors associated with obesity in SA. They analysed three national cross-sectional (repeated panel) surveys, using a multilevel logistic regression and population attributable fraction estimation which allowed for identification of contextualised high impact factors associated with obesity (BMI >30 kg/m^2) among adults (15 years+). They found that obesity prevalence increased significantly from 23.5% in 2008 to 27.2% in 2012, with a significantly (p <0.001) higher prevalence among females (37.9% in 2012) compared to males (13.3% in 2012). Living in formal urban areas, of white ethnicity, being married, not exercising and/or with higher SES, were significantly associated with male obesity. Females living in formal or informal urban areas, higher crime areas, African/White ethnicity, married, not exercising, with a higher SES and/or living in households with proportionate higher food expenditure (on unhealthy food options) were significantly more likely to be obese. The identified determinants appeared to account for 75% and 43% of male and female obesity, respectively. White males had the highest relative gain in obesity from 2008 to 2012. They concluded that the rising prevalence of obesity in SA is significant and over the 5-year period the rising prevalence of type 2 diabetes has mirrored this pattern, especially among females. Targeting young adolescent girls should be a priority. Addressing determinants of obesity will involve a multifaceted strategy at individual and population levels. With rising costs in the private and public sector to combat obesity-related NCD, this analysis can inform culturally sensitive mass communications and wellness campaigns. Knowledge of social determinants is critical to develop “best buys”.

In the present study, Peer et al. (2014)[118] found that among the 1099 participants, the calculated cut-off points for anthropometry were: males, WC 83.9 cm, WHR 0.89, waist-to-height ratio (WHtR) 0.50, BMI 24.1 kg/m^2; females, WC 94.0 cm, WHR 0.85, WHtR 0.59 and BMI 32.1 kg/m^2. Raised WC was significantly associated with >2 metabolic syndrome components in males: WC 84.0–93.9 cm OR: 3.19, 95% CI 1.73–5.85) and WC 94.0 cm compared to WC 84.0 cm, and in females: WC 80.0–93.9 cm and WC 94.0 cm compared to WC 80.0 cm. They also found that in the logistic model with BMI for females, obesity, but not overweight was significantly associated with >2 metabolic syndrome components. They concluded that obesity cut-off points for Africans should be re-evaluated and adjusted accordingly.

In addition, Peer et al. (2014)[118] also examined the prevalence and determinants of overweight/obesity in our population between 1990 and 2008/09. The mean BMI and WC were 23.7 kg/m^2 and 84.2 cm in males, and 33.0 kg/m^2 and 96.8 cm in females. Prevalence of BMI >25 kg/m^2 and raised WC were 28.9% in males, and 82.8% and 86.0% in females. Among 25–64-year-olds, BMI >25 kg/m^2 decreased between 1990 (37.3%) and 2008/09 (27.7%) in males but increased from 72.7% to 82.6% in females. In the regression models for males and females, higher BMI was directly associated with increasing age, wealth, hypertension and diabetes, but inversely related to daily smoking. They also found a rising BMI was significantly associated with raised LDL-C and being employed compared to unemployed in males, and having seven years’ education in females. They, thus, concluded that overweight/obesity, particularly in urban black females, requires urgent action as a result of the associations with CVD risk factors and their serious consequences.

Okop et al. (2015)[119] determined the factors associated with excessive body fat among black males and females living in rural and urban communities of SA. They investigated a cross-sectional analysis of data from the PURE study, Cape Town, conducted in 2009/2010. The study sample included 1220 participants (77.2% females) aged 35–70 years, for whom anthropometric measurements were obtained and risk factors documented through face-to-face interviews using validated international PURE study protocols. Sex-specific logistic regression models were used to evaluate socio-demographic, lifestyle and psychological factors associated with three excessive body fat indicators, viz. BMI, WC and BF%. They found that the prevalence of excessive body fat based on BF%, WC and BMI cut-offs were 96.0%, 86.1%, and 81.6% for females, respectively, and 62.2%, 25.9%, and 36.0% for males, respectively. The significant odds of excessive body fat among the currently married compared to unmarried were 4.1 (95% CI 1.3–12.5) for BF% and 1.9 (95% CI 1.3–2.9) for BMI among females; and 4.9 (95% CI 2.6–9.6), 3.2 (95% CI 1.6–6.4) and 3.6 (95% CI 1.9–6.8) for BF%, WC and BMI, respectively, among males. Age <50 years (compared to age >50 years) was inversely associated with excessive BF% in males and females, and less-than-a-college education was inversely associated with excessive BMI and WC in males. Tobacco smoking was inversely associated with all three excessive adiposity indicators in females, but not in males. Unemployment, depression, and stress did not predict excessive body fat in males or females. They concluded that the sex-differences in the socio-demographic and lifestyle factors associated with the high levels of excessive body fat in urban and rural females and males should be considered in packaging interventions to reduce obesity in these communities.

2.3 CONSEQUENCES OF URBANISATION

Massive urbanisation has taken place in South Africa from rural to urban areas in the past few decades, mainly due to the repeal of the law (in 1986) which had restricted movement of Africans. This has resulted in great demand on the existing urban resources in terms of health care, housing, water and electricity among others. Since the majority of migrants who come to the cities are mainly poor people, often without a very basic education, they may find it even more difficult to find work and may end up in a worse financial situation.[120] Hence urbanisation is also associated with poverty and low basic incomes for many in this situation. To a degree this also fuels the nutrition transition since poor people tend to buy cheap foods which are energy-dense and high in saturated fat, sugar and salt.

2.4 MAIN CHRONIC DISEASES

2.4.1 HEART DISEASE

Ischaemic heart disease is a multifaceted disease with numerous risk factors. Some of these risk factors are characteristics which are not within our control, e.g. sex, age and family history. However, some of the most important risk factors are those behaviours that we learned/acquired, e.g. smoking, a sedentary lifestyle, and poor dietary habits. Further to these are underlying conditions, such as hypertension, diabetes mellitus and hyperlipidaemia, especially hypercholesterolaemia.

The components of the diet and other aspects of lifestyle which are of the greatest importance in increasing the risk of CHD, are saturated fatty acids; trans-fatty acids, cholesterol, fibre, sugar, smoking, hypertension, lack of PA, and alcohol intake. These have been discussed under the modifiable risk factors.

Steyn et al.(1990)[121] assessed the CVD risk profile of the Coloured population of the WC (CRISIC). They reported that the IHD mortality rate (MR) for Coloureds was rapidly approaching that for whites and that among the females aged 35 years or older, the rates for Coloureds exceeded those for whites.

Ischaemic heart disease was the largest single cause of death in the Western Cape population, accounting for 12% of all deaths in 2000.[1] In addition to IHD, stroke, chronic obstructive pulmonary disease, diabetes mellitus and lung cancer were the five leading single causes of death, accounting for more than 50% of deaths.[1]

Alberts et al. (2005)[122] investigated the prevalence and associated risk factors of CVD in a rural adult black African population from Limpopo Province in SA. They found a high prevalence of tobacco use for males (57%) and females (35.4%), with 28.1% of the females using smokeless tobacco (snuff). Just under 52% of the females were either overweight or obese. Diabetes was diagnosed in 8.8% and 8.5% of females and males, respectively. High-density lipoprotein cholesterol was relatively high, whereas 42.3% of females and 28.5% of males had LDL-C levels of 3 mmol/l or more. Hypertension was found in 25.5% of females and 21.6% of males. According to the Framingham CVD risk score,18.9% of females and 32.1% of males had a 20% or higher chance of having a CVD event in the next ten years. Thus, there was a high prevalence of chronic disease risk factors and, consequently, this population had a higher than expected risk of developing a CVD event in the following ten years when compared to similar studies in black Africans.[122]

Pisa et al. (2012)[123] examined whether the association between SES and CVD risk factors in black South Africans from the North West Province had shifted from the more affluent groups, with higher SES, to the less affluent, lower SES groups over nine years. In 2005, cross-sectional baseline data of 2010 urban and rural subjects (35 years and older) participating in the PURE study were collected. These data were analysed to examine the relationship of level of education, employment and urban or rural residence with dietary intakes and other CVD risk factors.[123] These relationships were compared to those found nine years earlier in the THUSA study conducted in the same area. They found that urban females had higher BMI, serum TG and fasting glucose levels compared to rural females and that both urban males and females had higher BPs and followed a more westernised diet.[123] However, rural males and females had higher plasma fibrinogen levels. The more highly educated subjects (which included urban and rural subjects) were younger than those with no or only primary school education. Few of the risk factors differed significantly between education groups, except that more highly educated males and females had lower BMIs, and females had lower BP and TG levels. These females also followed a more prudent diet than those with only primary school education. Employed males and females had higher BMIs, higher energy intakes but lower plasma fibrinogen levels, and employed females had lower TG levels. No significant differences in total serum cholesterol values were observed. Pisa et al. (2012)[123] concluded that their results suggested a drift of CVD risk factors from groups with higher SES to groups with a lower SES from 1996 to 2005, indicating that interventions to prevent CVD should also be targeted at Africans living in rural areas, those with low educational levels, and the unemployed.[123]

2.4.2 STROKE

Bradshaw et al. (2000)[1] reported that stroke accounted for 2.8% of the premature mortality causes in 2000 in SA, with the Western Cape accounting for 4.6%.

Black African hypertensive patients in SSA are prone to cerebral haemorrhage, malignant hypertension leading to uraemia, and congestive heart failure, whereas coronary artery disease

(CAD) is relatively uncommon. Response to antihypertensive drugs like the beta-blockers and the angiotensin-converting enzyme (ACE) inhibitors are poor unless these agents are combined with a thiazide diuretic. Black African hypertensive patients respond best to diuretics, vasodilators or calcium channel blockers. A profiled approach to the treatment of hypertension in black patients is suggested. There is a paucity of representative dietary data available in SA on the black African population and, therefore, the results of the BRISK study together with those of the present study are vitally important if one is to have a chance of understanding the dietary patterns which influence the development of NCD in the black African population over time.

Maredza et al. (2015)[124] estimated the burden of stroke in rural SA using the epidemiological parameters of incidence, mortality and DALYs metric, a time-based measure that incorporates both mortality and morbidity. Data from the Agincourt health and socio-demographic surveillance system were utilised to calculate stroke mortality for the period 2007–2011. Dismod, an incidence-prevalence-mortality model, was used to estimate incidence and duration of disability in the Agincourt sub-district and 'mostly rural' municipalities of SA. Using these values, burden of disease in years of life lost (YLL), years lived with disability (YLD) and DALYs were calculated for the Agincourt sub-district. They found that over the five years, there were an estimated 842 incident cases of stroke in the Agincourt sub-district, a crude stroke incidence rate of 24 per 100 000 person years. They estimated that 1 070 DALYs are lost as a result of stroke annually. Of these, YLDs contributed 8.7% (3.5–10.5%) in sensitivity analysis). Crude stroke mortality was 114 per 100 000 person-years in 2007–2011 in the Agincourt sub-district. Burden of stroke in entire rural SA, a population of some 13 000 000 people, was high, with an estimated 33 500 strokes occurring in 2011. They concluded that this study provides the first estimates of stroke burden in terms of incidence, and disability in rural SA. High YLL and DALYs lost among the rural populations demand urgent measures for preventing and mitigating impacts of stroke. Longitudinal surveillance sites provide a platform through which a changing stroke burden can be monitored in rural SA.

2.4.3 CANCER

Cancer is a generic term for a large group of diseases that can affect any part of the body. Other terms used are malignant tumours and neoplasms. One defining feature of cancer is the rapid creation of abnormal cells that grow beyond their usual boundaries, and which can then invade adjoining parts of the body and spread to other organs, the latter process is referred to as metastasizing. Metastases are the major cause of death from cancer.[17]

The projection is that the number of people diagnosed with cancer will double by the year 2030, with most of the increase occurring in the middle- and low-income countries.[125] Many of the risk factors associated with cancer are modifiable, especially tobacco smoking, alcohol consumption, and poor diet. Owing to urbanisation and changing food systems, the prediction is that the prevalence of lifestyle-related cancers will continue to increase. This will result in a huge economic and social cost.

Cancers feature among the leading causes of morbidity and mortality worldwide, with approximately 14 million new cases and 8.2 million cancer-related deaths in 2012.[125] The number of new cases is expected to rise by about 70% over the next two decades. Among males, the five most common cancer sites diagnosed in 2012, were the lungs, prostate, colorectum, stomach, and liver. Among females, the five most common cancer sites diagnosed, were the breast, colorectum, lungs, cervix, and stomach. Around one third of cancer deaths are caused by the five leading behavioural and dietary risks, i.e. a high BMI, low fruit and vegetable intake, lack of PA, tobacco use, and alcohol use. Park et al. (2005)[126] evaluated the association between dietary fibre intake and risk of colorectal cancer. They concluded that in their large pooled analysis, dietary fibre intake was inversely associated with risk of colorectal cancer in age-adjusted analyses, however, after accounting for other dietary risk factors, high dietary fibre intake was not associated with a reduced risk of colorectal cancer.

Dahm et al. (2010)[127] also examined the association between dietary fibre intake and colorectal cancer risk, and conducted a prospective case-control study nested within seven UK cohort studies. They included 579 patients, who developed incident colorectal cancer, and matched them with 1996 control subjects. They found that intakes of absolute fibre and of fibre-intake density, ascertained by food diaries, were statistically significantly inversely associated with the risks of colorectal and colon cancers in both age-adjusted models and multivariable models that adjusted for age; anthropomorphic and socio-economic factors; and dietary intakes of folate, alcohol, and energy. However, no statistically significant association was observed when the same analysis was conducted using dietary data obtained by food-frequency questionnaire. They concluded that intake of dietary fibre is inversely associated with colorectal cancer risk.[127]

There has also been interest in whether intakes of specific types of fat are associated with breast cancer risk independently of other types of fat, but results have been inconsistent. Smith-Warner et al.(2001)[128] identified eight prospective studies and calculated relative risks. They found that no associations were observed for animal or vegetable fat intakes and that these associations were not modified by menopausal status. Their data were suggestive of only a weak positive association with substitution of saturated fat for carbohydrate consumption. None of the fats were significantly associated with breast cancer risk relative to an equivalent reduction in carbohydrate consumption.

Tobacco use is the most important risk factor for cancer causing around 20% of global cancer deaths and around 70% of global lung cancer deaths. Cancer-causing viral infections such as the hepatitis B virus (HBV), hepatitis C virus (HCV) and human papilloma virus (HPV) are responsible for up to 20% of cancer deaths in LMIC. More than 60% of world’s total new annual cases occur in Africa, Asia and Central and South America. These regions account for 70% of the world’s cancer deaths. Annual cancer cases are expected to rise from 14 million in 2012 to 22 within the next two decades. Cancer is also a leading cause of death worldwide, accounting for 8.2 million deaths in 2012. The most common causes of cancer deaths, are cancer of the lung (1.59 million), liver (745 000), stomach (723 000), colorectal (694 000), breast (521 000), and oesophageal cancer (400 000). Tobacco use, alcohol use, unhealthy diet, and physical inactivity are the main cancer risk factors worldwide. Some chronic infections are risk factors for cancer and have major relevance in LMIC. The hepatitis B virus, HCV and some types of HPV increase the risk for liver and cervical cancer, respectively. HIV infection substantially increases the risk of cancer, such as cervical cancer.[125]

2.4.4 TYPE 2 DIABETES

While communicable diseases, such as HIV/AIDS, malaria, and tuberculosis have continued to pose greater threats to the public health system in SSA, it is now apparent that NCD, such as diabetes mellitus, are undoubtedly adding to the multiple burden people in this region are suffering.[129] Furthermore, type 2 diabetes is the most common form of diabetes (90–95%), exhibiting an alarming prevalence among people of this region. Main risk factors include obesity, rapid urbanisation, physical inactivity, ageing, nutrition transition, and socio-economic changes. Patients in SSA also show manifestations of β-cell dysfunction and insulin resistance.

However, because of strained economic resources and a poor healthcare system, most patients are diagnosed only after they have overt symptoms and complications of diabetes. Microvascular complications are the most prevalent, but metabolic disorders and acute infections cause significant mortality. The high cost of treatment of type 2 diabetes and its comorbidities, the increasing prevalence of its risk factors, and the gaps in the healthcare system, necessitate that solutions be planned and implemented urgently. Aggressive actions and positive responses from well-informed governments appear to be needed for the conducive interplay of all forces required to curb the threat of type 2 diabetes in SSA.

Obesity is also known to be a modifiable risk factor for the development of type 2 diabetes. In the African-American population, obesity and diabetes were assessed, and not only was the diabetes problem found to be disproportionately large but also obesity was very high, particularly in females.[130] The investigators speculated about the possibility that African-Americans descended from African blacks~~,~~ could share a gene pool that makes them more susceptible to diabetes and also more severely affected once they have it. However, the current data from Africa does not support this notion.

The diabetes prevalence in Africa generally has been reported as being lower than in African-Americans, although in more urban areas of SA some have reported similar prevalences.[130] The prevalence of type 2 diabetes and associated risk factors in urban black Africans in Cape Town was determined and found to be moderately high. This is considerably higher than previous reports from Africa and the association of type 2 diabetes with urbanisation has important implications in view of the large-scale urbanisation occurring in Southern Africa.[131]

Diabetes was in the top 20 leading single causes of death in the entire Western Cape population, accounting for 3.8% of deaths, with females having a higher number of deaths than their male counterparts.[1]

In the 2003 SADHS, the self-reported prevalence of diabetes in urban males was found to be 2.9% and urban females 4.4% compared to 1.7% and 2.7% in the rural males and females, respectively.[114] In the SANHANES study, 8.2% of black Africans had HbA1c levels >6.0% and the self-reported diabetes rate was 5.3% in black African females and 3.0% in males.[8]

In the present study Peer et al. (2012)[132] examined the prevalence of diabetes and compared this to that of the BRISK study done in 1990. They found the prevalence of diabetes to be 13.1%, which peaked in 65–74-year-olds (38.6%). Among the diabetic participants, 57.9% were known and 38.6% treated. Using the 1985 WHO criteria, age-standardised diabetes prevalence was higher by 53% in 2008/09 (12.2% (10.2–14.2)) compared to 1990 (8.0% (5.8–10.3)). They concluded that the current high prevalence of diabetes in urban-dwelling South Africans, and the likelihood of further rises given the high rates of obesity, is concerning. Multi-facetted diabetes prevention strategies are essential to address this burden.

CHAPTER 3: METHODS

3.1 RESEARCH QUESTIONS

Did the dietary intake of black adults residing in Cape Town townships change significantly between 1990 and 2009 with respect to energy and nutrient intakes, which are commonly associated with the nutrition transition (energy, macronutrients, sugar and fibre).

Are energy and nutrient intakes associated with anthropometric, biochemical, BP, urbanisation and assets (SES) of participants in 2009?

3.2 AIMS

To determine the dietary intake of the urban black population (25–64 years) of Cape Town in 2009 and compare these findings with a similar sample examined in the same townships in 1990.

3.3 SPECIFIC OBJECTIVES

i) To determine the dietary intake (macronutrients and micronutrients), number of food portions, and meal pattern (meals and snacks) of black adults living in Cape Town in 2009.

i) To determine the dietary intake of black adults living in Cape Town in 2009 and to compare this data with dietary data from 1990.

ii) To determine the anthropometric data (BMI, WC, HC, WHR) and BP of black adults in Cape Town in 2009.

iii) To determine the association between dietary intake with anthropometric data: BMI, WC, HC, WHR; and BP of black adults in Cape Town in 2009.

iv) To determine the association between dietary intake with biochemical data: TC, TG, HDL-C, LDL-C levels and glucose of black adults in Cape Town in 2009.

vii) To determine the association between dietary intake with duration of urbanisation (years lived in the city) of black adults in Cape Town in 2009.

viii) To determine the association between dietary intake with an asset index (measure of SES) of black adults in Cape Town in 2009.

3.4 HYPOTHESES

3.4.1 There are no significant differences in dietary intake (energy, macro-, micronutrients, food groups and food portions) data of black adults in 2009 compared to 1990.

3.4.2 There are no significant differences in association between energy and nutrients with urbanisation and assets of black adults in 2009. (The published data from1990 does not include comparative data).

3.4.2 There are no significant associations among dietary intake data with anthropometry (BMI, WC, HC, WHR), BP, biochemical data (TC, HDL-C, LDL-C, TG and glucose); and BP of black adults in 2009. The primary investigator (Nasheeta Peer) has published the anthropometric and biochemical data comparisons with 1990 data)

3.5 STUDY METHODOLOGY

3.5.1 STUDY DESIGN

A comparison of results from two cross-sectional studies undertaken in the black urban population in the same townships, twenty years apart (1990 and 2009).

3.5.2 STUDY POPULATION

The study population comprises urban black Africans between ages 25 and 74 years, who reside in the Cape Town metropolitan suburbs of Langa, Gugulethu, Khayelitsha, Nyanga, and Crossroads. Data collection started in 2008 and continued until the end of 2009.

3.5.3 STUDY SAMPLE

This dietary study forms part of a larger study that was done to determine the prevalence of diabetes in black adults in Cape Town.[132] The sample size was determined according to an estimated diabetic prevalence of 8% in this population. The prevalence of diabetes was used since this was one of the main objectives of the larger study. Thus, with a sample size of 1260, the 95% CI for the expected prevalence of diabetes of 8% will extend 0.015 from the observed prevalence.

3.5.4 SAMPLING AND SAMPLING FRAME

The sampling frame was based on the BRISK study conducted in 1990 and included participants living in the townships of Langa, Gugulethu, Khayelitsha, Crossroads, and Nyanga. There was proportionate sampling of these areas according to their population density.

An aerial map of each township which showed the houses, was used to randomly select the households. Individuals from these households were selected on a quota for age and gender categorisation; the rationale being that the risk factors and disease prevalence differ among age groups and genders. There is a minimum of 100 participants in each of the 10 strata, stratified for age (25–34, 35–44, 45–54, 55–64, and 65–74) and sex (male and female).

3.5.5 EXCLUSIONS

Exclusions were made for the following adults:

- pregnant and lactating females;
- those who were bedridden;
- those unable to give consent;
- those on tuberculosis treatment;
- those on antiretroviral therapy;
- cancer patients having received treatment within the last year; and
- individuals residing in Cape Town for less than 3 months.

3.5.6 REFUSALS AND NON-RESPONDERS

Replacements were allowed when:

- confronted with individuals not suitable for participation, as set out in the exclusion criteria above;
- faced with refusals;
- being unsuccessful in contacting the randomly selected participant from a randomly selected household after the third failed attempt.

3.5.7 REPLACEMENTS

The field worker was expected to replace the individual with another participant from the same household. Failing that, the field worker approached the household immediately to the left for a replacement participant. Substitutions and refusals were recorded.

3.5.8 ETHICAL APPROVAL

Ethical approval for the main study was granted by the Ethics Committee of the University of Cape Town (REC REF: 224/2006). Ethical approval was also granted from the ethics committee of the University of Stellenbosch for the analyses of the dietary and nutritional status data of the main study for this Master's degree in Nutrition. When the main study was conducted, participants received a detailed explanation of the study procedure in Xhosa, their primary language. This was followed by written informed consent from each participant in the study and confidentiality was ensured. Participants were posted their blood results and a referral letter to the appropriate health services was included if clinically relevant abnormalities were identified by the attending doctor (NP). Participant information remained confidential in the analysis of data in the current study, and only the researcher and study leaders had access to their names. Participants were allocated an identification number which was used on all documents. Questionnaires and other data were stored at the MRC for 5 years under lock and key.

3.6 DATA COLLECTION, MANAGEMENT AND METHODS OF INTERPRETATION

All data collection was done by trained and registered Xhosa speaking nurses employed by the MRC. They also have research experience having participated in prior dietary research studies. All measurements and taking of blood was done in central clinics in the townships surveyed.

3.6.1 SOCIO-DEMOGRAPHIC DATA

Participants were classified by age, level of education and type of housing (Questionnaire Addendum 4). Urbanisation was calculated based on years of life lived in the city using questions 2e and 2f of the questionnaire. The asset index was calculated from question 3f in Addendum 4. All assets were added for each individual up to a total of 11. Total scores of all the sample were then classified into tertiles with the poorest falling within the first tertile and the "richest" falling in the highest tertile. The questions about employment status were obtained from questions 3a to 3e.

3.6.2 ANTHROPOMETRY

Weight was measured to the nearest 0.1 kg with the subject wearing light clothing and standing barefoot on a portable scale that was calibrated regularly. Height was measured to the nearest 0.1 cm with the participant standing barefoot using a stadiometer. The waist measurement was taken at the smallest circumference between the xiphi sternum and the umbilicus on expiration. Measurements were taken to the nearest 0.1 cm after normal expiration while in an upright position.[133] Hip measurements were taken to the nearest 0.1 cm at the maximum posterior protuberance of the buttocks with the participant standing upright with feet together.[133] Body mass index was calculated as the individual's weight in kilograms divided by their height in metres squared (kg/m²). Overweight is defined as BMI 25–29.9 kg/m^2 and obesity as BMI ≥30 kg/m^2. Underweight is defined as a BMI <18.5 kg/m².[134] Waist circumference per se will be assessed for abdominal obesity. The latest IDF cut-off points for WC will be used, ≥80 cm for females and ≥94 cm for males.[135] BMI categories were as follows:[134]

- Underweight = BMI <18.5 kg/m^2
- Normal = BMI 18.5 to 24.9 kg/m^2
- Overweight = BMI 25 to 29.9 kg/m^2
- Obese Class I = BMI 30 to 35 kg/m^2
- Obese Class II = BMI 35.1 to 40 kg/m^2
- Obese Class III = BMI 40.1 to 45 kg/m^2
- Morbid obesity = BMI >45 kg/m^2.

3.6.3 DIETARY INTAKE

Data collection was done by means of a single 24HR. This method was used based on the fact that the 1990 study also used a single 24HR.[34,136] The 24HR is a retrospective method (the subject has to think back) to find out what he/she had had to eat/drink. The method involves asking the subject what he/she had eaten/drank during the previous 24 hours. One usually starts the 24-hour period at the point that the subject woke up the previous day. Food/drink intake is then recalled until the same time on the day of the interview. Since it is not always so easy to remember exactly what one eats/drinks, the interviewer has to lead the subject very carefully through the day to help him/her remember all the detail.

The multiple pass method was used to do the 24HR.[137] Step 1 involves getting a general idea of what the person ate/drank. In Step 2 you help the person to remember anything that he/she might have forgotten by going through a list of items that are often forgotten. In Step 3 you are going to find out the following detail about each food item/drink identified in Steps 1 and 2: the time that it was eaten/drank, a detailed description of the item, and its preparation and portion size. The photo manual is used to do this.[138] The last step (Step 4) involves a last attempt to identify anything that the subject might have forgotten.

To determine the dietary quality, the values of macro- and micronutrients were compared to the DRI including the RDA, the EAR, adequate intakes (AI) and the acceptable macronutrient distribution ranges (AMDR).[139] The latter provided information on acceptable dietary ranges for CHO, fat and protein as a percentage of energy intake. Dietary quality was also assessed by determining the nutrient adequacy ratio (NAR) of each micronutrient.[140] This was based on the mean percentage of the EAR value of the nutrient. For example, if a nutrient had a mean NAR of 75% then it met the EAR value by 75%. A 100% met the EAR value completely. The mean adequacy ratio (MAR) of the diet comprised the sum of the NAR values, truncated to 100%, divided by 11.

Food groups were calculated based on the same groups selected in the 1990 study, namely dairy, meat, fruit and vegetables, cereals and fats. The contribution of these groups to energy intake, fat, cholesterol and protein intake was calculated. Additionally the number of daily meals were calculated for each participant as well as the percent who consumed breakfast, lunch and dinner.

3.6.4 BIOCHEMISTRY

3.6.4.1 Standard oral glucose tolerance test

After an overnight fast of 8–14 hours, an indwelling cannula was inserted into a vein in the antecubital fossa. A fasting blood sample was drawn and further blood samples were taken at 30 and 120 minutes after the participant drank 75 g of anhydrous glucose in 250–300 ml of water over the course of 5 minutes. The timing of the test was from the beginning of the drink. Blood samples were collected 2 hours after the test load.[141] Impaired glucose tolerance (IGT) is defined as plasma glucose between

7.8 mmol/l and 11.1 mmol/l, 2 hours' post-glucose load. Impaired fasting glycaemia is fasting plasma glucose between 6.1 mmol/l and 7.0 mmol/l.[141]

3.6.4.2 Lipid profile

A fasting blood sample was taken to ascertain TC, HDL-C, LDL-C and TG levels. Lipid profile abnormalities included a TC >5 mmol/l, TG >1.5 mmol/l, HDL-C <1.2 mmol/l, and calculated LDL-C >3 mmol/l and HDL-C/TC ratio <20% (South African Medical Association and Lipid and Atherosclerosis Society of Southern Africa Working Group, 2000).[142] Once drawn, blood samples were placed in the appropriate tubes and kept on ice. They were delivered to the laboratory within six hours where the blood was spun and centrifuged. The blood samples were analysed for glucose, insulin, creatinine and lipid levels at the University of Cape Town's laboratory.

3.6.5 BLOOD PRESSURE

Blood pressure measurements were in compliance with the AHA recommendations using an automated Omron BP M6 Comfort Machine. Blood pressure was measured three times at five minute intervals on the participant's left arm, while seated and at rest. The mean of the second and third readings were used in this analysis. The Omron used, had a single integrated cuff (one size fits all). Subjects were asked to refrain from smoking and caffeine consumption for 30 minutes prior to the measurements. The cut-off points for high BP used, was 140/90 mmHg.[78]

3.7 DATA ANALYSIS AND STATISTICAL INTERPRETATION

3.7.1 DATA ANALYSIS

In this study, data will not be presented on the biochemical and BP results, since these have been part of a larger study and have been published.[84,132] However, associations among these data will be tested with the dietary data.

The final sample of 1119 dietary questionnaires were checked, entered and cleaned by the investigator (Nasreen Jaffer) over nine months.

Anthropometric data were calculated according to the CDC/World Health Organization Anthro' package, which is regarded as the standard programme for such analysis. Analysis relating to calculation of dietary data utilizing the SAMRC Food Tables were done on a set of programmes developed by the Institute of Biostatistics at the SAMRC. These programmes are summarised in a report by Langenhoven et al. (1991)[143]

The dietary data were coded, and the South African Food Composition Tables (2004) used to calculate the dietary intake of every person and data were adjusted for fortification values. Basic descriptive univariate statistical analyses were performed in the exploratory stage of the data analysis. In order to compare dietary data with the 1990 data means and standard deviations were calculated.

Further bivariate analyses of dietary variables against various socio-demographic variables also took place. In addition, age and gender were usually reported along with these bivariate analyses. Significance testing was performed for different nutrients between the CRIBSA (2009) and the BRISK (1990) studies. Predictive models of dietary consumption against socio-demographic and other variables potentially influencing the reporting of dietary intake was performed by multiple linear regression. Correspondence analysis was also used to elucidate the multivariate relation between various dietary, socio-demographic and degenerative disease risk-factor variables.

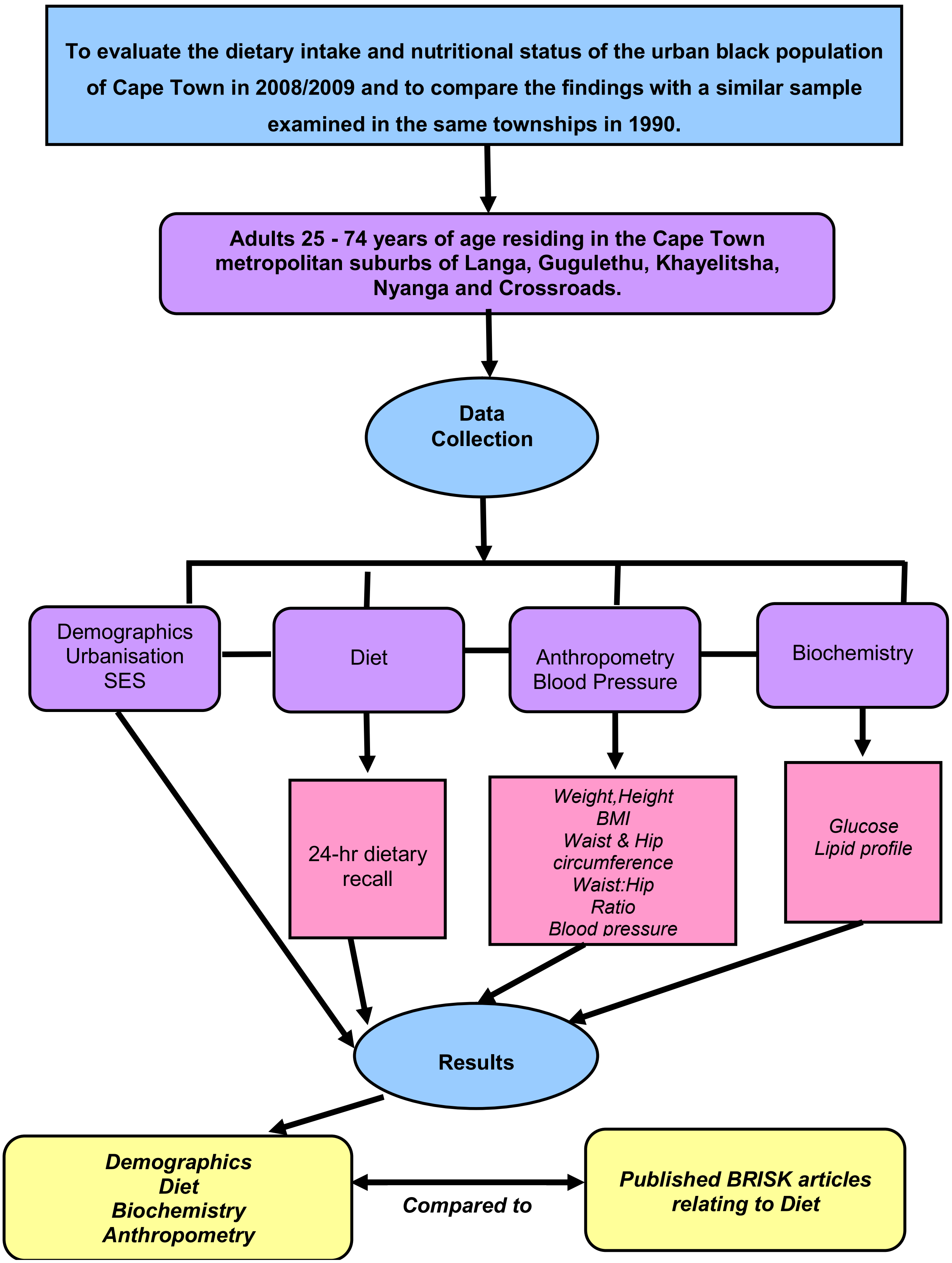
A principal component analysis of the pooled data, based on the assets that defined wealth, was used to develop an asset index.[144] This was categorized into tertiles with relative wealth classified as poorest, poor and least poor. In lieu of the absence of an internationally agreed definition on what constitutes an urban environment, the proportion of life spent in the city, used in previous studies, defined the degree of urbanisation.[145] Length of urban residence was recorded by summing the duration lived in a city, from birth until the date of data collection. We further computed a linear regression model using MAR as the dependent variable and added age, gender, urbanisation, asset index and other variables to the model. Correlations between the asset index and urbanisation duration were done with energy and nutrient intakes using Pearson's correlations. Regressions were done to test the significance of various variables.

3.7.3 FEEDBACK TO THE COMMUNITY

Throughout the contact-making phase, assurances had to be provided that ways would be sought to give feedback to the areas surveyed and to the African population as a whole. There were many conflicting opinions as to how this should be done, especially when the issue of confidentiality arose. Furthermore, practical considerations had to be taken into account. For example, those residing in the squatter camps frequently had no formal addresses. Finally, it was left to individual respondents to respond to the offer of feedback by providing addresses of friends, relatives or employers, to whom results could be posted.

After completing the data capturing and cleaning on computer, the first priority was to give feedback to respondents in various ways. A result sheet was developed by the Institute for Biostatistics (SAMRC). A letter explaining the results was drawn up and translated in Xhosa, as well as an information sheet on a healthy lifestyle to protect against heart disease (Appendix I). An address list was computerised and merged with the result by ID number. Of the 799 result sheets sent out, about 130 were returned by post due to unknown addresses. The BRISK study however, received publicity via Radio Xhosa and respondents who had not received their results were given a contact number that could be used to request them.

3.8 CONCEPTUAL FRAMEWORK



CHAPTER 4: RESULTS

4.1 SOCIO-DEMOGRAPHIC DATA

The sampling procedure yielded a study population of 1099, with 392 males and 707 females who have been divided into four categories representing young adults, mid-life, older and elderly groups. The results are presented in categories according to a level of education, employment status, type of housing according to the socio-demographic characteristics found in Section 3 of the questionnaire. Degree of urbanisation was categorised from Section 2 on the migratory history into years of life spent in a city. The asset index was calculated from question 3F, which gave an indication of the number of assets per household.

Table 4.1 presents data on the sample which includes 392 males and 707 females (n=1099). Most adults had an education of at least 8–12 years. A large per cent of males (60%) and females (58.3%) were unemployed, and 13.8% were pensioners. Twenty-one percent of adults lived in formal houses, 35.4% in council houses or hostels and 43.6% in shacks. Only 2.1% of adults had spent less than 20% of their life in an urban area. Adults were more or less equally spread among tertiles of the asset index.

Table 4.1: Descriptive statistics of the socio-demographics of the study population of the urban black population in the Cape Town in 2009

	Males n=392	Females n=707	All n=1099	*P*-value
Age (%)				
25–34	27.4	29.9	29.0	0.6977
35–44	26.9	23.9	25.0	
45–64	37.9	38.4	38.2	
65–74	7.9	7.8	7.8	
Education (%)				
None	6.9	6.5	6.6	0.2421
1–7 years	32.7	30.4	31.2	
8–12 years	53.6	58.6	56.8	
Higher (tertiary or diploma)	6.9	4.5	5.4	
Employment status (%)				
Employed	23.2	29.9	21.1	0.1272
Unemployed	60.0	58.3	58.9	
Other: homemaker	1.0	1.6	1.4	
Pensioners	10.2	15.8	13.8	
Disabled	4.9	3.7	4.1	
Students	0.8	0.7	0.7	
Type housing (%)				
Built formal unit (private)	22.2	20.4	21.0	0.0066**
Council/core house/hostel	29.3	38.8	35.4	
Informal shack	48.5	40.9	43.6	
% Life in urban area				
≤20%	11.2	12.6	12.1	0.1457
21–69.9%	43.4	48.1	46.4	
≥70%	45.4	39.3	41.5	
Asset Index by tertiles				
1st (poorest)	142 (36.2)	221 (31.3)	363 (33.0)	0.0262*
2nd	115 (29.3)	264 (37.3)	379 (34.5)	
3rd (richest)	135 (34.4)	222 (31.4)	357 (32.5)	

* Chi square test $p < 0.05$; ** Chi square test $p < 0.01$.

4.2 ANTHROPOMETRY

The anthropometric data of males are presented in Table 4.2.1a. In terms of mean weight, height, WC, and HC the youngest two age categories were generally shown to be significantly lower than those of the two older age categories. The two youngest age categories had mean BMI values in the normal range (18.5–24.9 kg/m^2), while the two older age categories were in the overweight category (25–29.9 kg/m^2).

In females, the mean weight, BMI, WC, and HC were significantly lower in the youngest two age groups (Table 4.2.1b). In all age categories mean BMI values were in the obese category (BMI≥30 kg/m^2). With regard to education it was found that mean weight, BMI, and HC were significantly different between those with no formal education and those with tertiary education, with the latter having the highest mean values. There were significant differences in weight, BMI, WC and WHR between those living in formal houses and those living in shacks, with the latter having the lowest mean values. Similarly, those in the richest group had significantly higher mean weights, BMI, WC and HC than those in the lowest tertile.

Table 4.2.2a presents data on BMI categories for males. Overall, 11.3% had a BMI <18.5 kg/m^2, 49.1% had a BMI between 18.8–24.9 kgm^2, 27.6% had a BMI 25–29.9 kg/m^2 (overweight), and 12% had a BMI ≥30 kg/m^2 (obese). There was a significant relationship between BMI and age (p <0.01); BMI and type of housing (p <0.05); and between BMI and the asset index (p <0.0001). In females, 1.3% had a BMI <18.5 kg/m^2, 11.6% had a BMI between 18.5–24.9 kg/m^2, 24.2% had a BMI 25–29.9 kg/m^2 (overweight), and 63% had a BMI ≥30 kg/m^2 (obese) (Table 4.2.2b). There was a significant relationship between BMI and age (p <0.01), and between BMI and the asset index, with those in the highest tertile having the highest BMI (p <0.0001).

Table 4.2.1a: Anthropometric data of the male urban black population (mean and SD) in Cape Town by age, education level, employment status, housing, urbanisation and asset index

	N	Weight (kg)		Height (m)		BMI (kg/m^2)		WC (cm)		HC (cm)		WHR	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
All subjects	391	69.64	15.70	1.70	0.07	24.01	5.26	85.72	13.80	95.67	10.26	0.89	0.08
Age (years)													
25–34	107	64.94 [B]	10.40	1.72	0.07	22.10 [B]	3.52	78.16 [B]	8.85	92.81 [B]	7.83	0.84 [D]	0.05
35–44	105	69.17 [A;B]	15.00	1.71	0.08	23.61 [A;B]	4.68	83.41 [B]	11.53	95.38 [A;B]	9.92	0.87 [C]	0.06
45–64	148	72.47 [A]	18.05	1.69	0.06	25.31 [A]	6.06	90.85 [A]	14.51	97.46 [A]	11.60	0.93 [B]	0.07
65–74	31	73.98 [A]	17.79	1.69	0.06	25.77 [A]	5.95	95.18 [A]	16.15	98.01 [A]	10.02	0.97 [A]	0.09
Education													
None	27	68.76	12.01	1.70	0.08	23.73	3.74	88.37	11.07	95.21	8.71	0.93 [A]	0.07
1–7 years	128	70.49	17.53	1.70	0.06	24.37	5.87	87.49	15.20	95.83	11.53	0.91 [A;B]	0.08
8–12 years	210	68.86	14.99	1.70	0.07	23.71	4.98	84.32	13.29	95.32	9.73	0.88 [B]	0.07
Higher (tertiary)	27	72.57	15.72	1.71	0.07	24.93	5.66	85.73	12.30	98.08	9.45	0.87 [B]	0.07
Employment status													
Employed	91	72.77	16.21	1.69	0.08	25.48	5.24	87.70 [A;B]	13.48	98.48	11.09	0.89 [A;B]	0.07
Unemployed	235	67.50	14.97	1.71	0.07	23.04	4.99	83.38 [A;B]	12.88	94.25	9.84	0.88 [A;B]	0.07
Other: homemaker	4	66.7	1.61	1.68	0.10	23.86	3.04	83.88 [A;B]	11.59	91.75	1.50	0.91 [A]	0.12
Pensioners	39	74.96	17.71	1.68	0.06	26.38	6.04	94.80 [A]	16.27	98.25	10.55	0.96 [A]	0.09
Disabled	19	72.63	16.25	1.71	0.05	24.75	4.80	89.36 [A]	12.45	95.43	8.97	0.93 [A]	0.07
Students	3	58.07	5.66	1.68	0.04	20.39	1.19	70.60 [B]	2.75	87.83	1.46	0.80 [B]	0.02
Housing type													
Built formal unit	87	73.97 [A]	18.03	1.71	0.07	25.30 [A]	5.98	89.03 [A]	16.06	98.31 [A]	11.22	0.90 [A;B]	0.09
Council/core house/hostel	115	72.27 [A]	16.13	1.71	0.07	24.67 [A]	5.72	88.36 [A]	13.41	97.30 [A]	10.47	0.91 [A]	0.07
Informal shack	190	66.08 [B]	13.47	1.69	0.07	23.02 [B]	4.40	82.63 [B]	12.21	93.49 [B]	9.21	0.88 [B]	0.07
Urbanisation													
<20%	43	65.76	15.46	1.69	0.07	22.96	4.85	82.39	13.23	93.69	9.27	0.88	0.07
20–69.9%	170	69.87	15.83	1.70	0.66	24.12	5.26	86.12	13.48	95.80	10.33	0.90	0.08
≥70% in urban	178	70.36	15.65	1.71	0.08	24.16	5.36	86.15	14.19	96.03	10.42	0.89	0.08
Asset index by tertiles													
1st (poorest)	141	64.06 [C]	13.06	1.69 [B]	0.07	22.40 [B]	4.50	81.14 [C]	11.47	92.15 [B]	9.53	0.88 [B]	0.07
2nd	115	70.15 [B]	15.01	1.72 [A]	0.07	23.83 [B]	5.08	85.69 [B]	13.24	96.32 [A]	9.92	0.89 [B]	0.07
3rd (richest)	135	75.04 [A]	16.97	1.70 [A;B]	0.07	25.85 [A]	5.59	90.54 [A]	14.91	98.80 [A]	10.21	0.91 [A]	0.08

BMI: body mass index; WC: waist circumference, HC: hip circumference; WHR: waist-hip ratio; SD: standard deviation; [A], [B], [C]: different symbols indicate significant difference between groups, Bonferroni multiple comparisons, *p* <0.05.

Table 4.2.1b: Anthropometric data of the female urban black population (mean and SD) in Cape Town by age, education level, employment status, housing, urbanisation and asset index

	N	Weight Mean	(kg) SD	Height Mean	(m) SD	BMI Mean	(kg/m^2) SD	WC Mean	(cm) SD	HC Mean	(cm) SD	WHR Mean	SD
All subjects	706	84.01	21.09	1.59	0.07	33.12	8.12	97.46	15.02	114.78	15.59	0.85	0.07
Age (years)													
25–34	211	79.59 [B]	19.05	1.60 [A]	0.06	31.00 [B]	7.47	92.11 [B]	14.80	111.90 [B]	13.42	0.82 [B]	0.07
35–44	169	85.39 [A;B]	21.89	1.60 [A]	0.07	33.19 [A;B]	8.25	97.01 [A;B]	14.65	114.43 [A;B]	15.69	0.85 [A]	0.07
45–64	271	86.81 [A]	21.73	1.59 [A]	0.07	34.46 [A]	8.24	101.17 [A]	14.25	116.76 [A;B]	16.69	0.87 [A]	0.08
65–74	55	82.98 [A;B]	20.55	1.55 [B]	0.06	34.51 [A]	8.08	101.11 [A]	14.70	117.18 [A]	16.02	0.86 [A]	0.06
Education													
None	46	77.10 [B]	22.14	1.59	0.07	30.49 [B]	7.81	94.36	11.74	109.76 [B]	16.64	0.87	0.07
1–7 years	215	83.31 [A;B]	22.36	1.59	0.08	33.02 [A;B]	8.27	98.19	15.64	113.84 [A;B]	16.75	0.86	0.08
8–12 years	414	84.62 [A;B]	20.02	1.59	0.06	33.33 [A;B]	7.96	97.17	14.83	115.53 [A;B]	14.81	0.84	0.07
Higher (tertiary)	32	90.83 [A]	22.37	1.62	0.06	34.96 [A]	9.18	100.82	17.01	118.69 [A]	14.45	0.85	0.09
Employment status													
Employed	141	83.52	20.99	1.60	0.06	32.80	8.41	95.67	14.57	114.37	14.69	0.84	0.07
Unemployed	411	83.79	21.19	1.60	0.07	32.82	7.95	96.90	15.31	114.11	15.39	0.85	0.08
Other: homemaker	11	79.39	19.93	1.63	0.07	30.14	7.92	93.75	13.91	109.03	14.64	0.86	0.08
Pensioners	112	85.76	20.73	1.57	0.07	34.96	8.21	101.74	14.16	118.50	17.04	0.86	0.07
Disabled	26	83.00	23.31	1.60	0.07	32.34	8.42	98.65	14.69	112.62	16.11	0.88	0.06
Students	5	92.50	20.46	1.59	0.04	36.70	7.93	99.68	14.68	121.48	13.62	0.82	0.06
Housing type													
Built formal unit	144	87.04 [A]	19.45	1.59	0.06	34.41 [A]	7.33	99.78 [A]	13.26	115.89	14.76	0.86 [A]	0.08
Council/core house/hostel	274	84.73 [A;B]	21.14	1.60	0.07	33.40 [A;B]	8.46	97.54 [A;B]	14.55	116.27	15.43	0.84 [B]	0.07
Informal shack	289	81.83 [B]	21.66	1.59	0.07	32.23 [B]	8.10	96.23 [B]	16.16	112.82	15.99	0.85 [A;B]	0.08
Urbanisation													
<20%	89	81.07	20.75	1.60	0.06	31.74	7.92	94.26	15.13	112.38	14.55	0.84	0.08
20–69.9%	340	84.27	20.72	1.59	0.07	33.17	7.79	97.93	14.81	114.92	15.21	0.85	0.08
≥70% in urban	278	84.63	21.65	1.59	0.07	33.50	8.56	97.90	15.18	115.37	16.35	0.85	0.07
Asset index by tertiles													
1st (poorest)	221	77.23 [C]	18.87	1.59	0.07	30.49 [C]	7.26	92.95 [B]	14.01	109.70 [C]	13.85	0.85	0.07
2nd	264	84.94 [B]	22.10	1.59	0.07	33.48 [B]	8.48	98.28 [A]	16.01	115.53 [B]	16.49	0.85	0.08
3rd (richest)	222	89.67 [A]	20.17	1.59	0.07	35.32 [A]	7.80	100.96 [A]	13.68	118.94 [A]	14.78	0.85	0.08

BMI: body mass index; WC: waist circumference, HC: hip circumference; WHR: waist hip ratio; SD: standard deviation; [A], [B], [C]: different symbols indicate significant difference between groups, Bonferroni multiple comparisons, p <0.05.

Table 4.2.2a: Mean BMI (kg/m²) and BMI categories of black males in Cape Town by age, education level, employment status, housing, urbanisation and asset index

	N	Mean BMI	SD	BMI <18.5	BMI 18.5–24.9	BMI 25–29.9	BMI 30–34.9	BMI 35–39.9	BMI ≥40
All subjects	391	24.01	5.26	11.3	49.1	27.6	8.7	1.5	1.8
Age (years)**									
25–34	107	22.10 [B]	3.52	34.1	36.5	16.7	11.8	0.0	0.0
35–44	105	23.61 [A;B]	4.68	29.6	25.5	30.6	23.5	16.7	14.3
45–64	148	25.31 [A]	6.06	31.8	31.8	44.4	47.1	66.7	71.4
65–74	31	25.77 [A]	5.95	4.6	6.3	8.3	17.7	16.7	14.3
Education									
None	27	23.73	3.74	2.3	7.8	7.4	5.9	0.0	0.0
1–7 years	128	24.37	5.87	36.4	30.7	30.6	41.2	50.0	42.9
8–12 years	210	23.71	4.98	61.4	53.1	55.6	44.1	50.0	42.9
Higher (tertiary)	27	24.93	5.66	0.0	8.3	6.5	8.8	0.0	14.3
Employment status*									
Employed	91	25.48	5.24	11.4	17.7	35.2	32.4	33.3	14.3
Unemployed	235	23.04	4.99	77.3	69.3	44.4	38.2	50.0	57.1
Other: homemaker	4	23.86	3.04	0.0	1.0	1.9	0.0	0.0	0.0
pensioners	39	26.38	6.04	6.8	6.3	13.0	20.6	16.7	28.6
disabled	19	24.75	4.80	4.6	4.2	5.6	8.8	0.0	0.0
students	3	20.39	1.19	0.0	1.6	0.0	0.0	0.0	0.0
Housing type*									
Built formal unit	87	25.30 [A]	5.98	15.9	19.3	26.9	26.5	16.7	57.1
Council/core house/hostel	114	24.67 [A]	5.72	29.6	24.0	35.2	32.4	66.7	28.6
Informal shack	190	23.02 [B]	4.40	54.6	56.8	38.0	41.2	16.7	14.3
Urbanisation									
<20%	44	22.96	4.85	13.6	13.0	5.6	14.7	16.7	0.0
20–69.9%	170	24.12	5.26	45.5	40.1	50.0	41.2	33.3	42.9
≥70% in urban	178	24.16	5.36	40.9	46.9	44.4	44.1	50.0	57.1
Asset index by tertiles***									
1st (poorest)	141	22.40 [B]	4.50	52.3	44.8	23.2	14.7	16.7	14.3
2nd	115	23.83 [B]	5.08	34.1	27.1	30.6	32.4	33.3	28.6
3rd (richest)	135	25.85 [A]	5.59	13.6	28.1	46.3	52.9	50.0	57.1

BMI: body mass index; WC: waist circumference, HC: hip circumference; WHR: waist hip ratio; SD: standard deviation; [A], [B], [C]: different symbols indicate significant difference between groups, Bonferroni multiple comparisons, p <0.05; * Significant relationship with BMI group, Chi square test, p <0.05; ** Significant relationship with BMI group, Chi square test, p <0.01; *** Significant relationship with BMI group, Chi square test, p <0.0001.

Table 4.2.2b: Mean BMI (kg/m^2) and BMI categories of black females in Cape Town by age, education level, employment status, housing, urbanisation and asset index

	N	Mean BMI	SD	BMI <18.5	BMI 18.5–24.9	BMI 25–29.9	BMI 30–34.9	BMI 35–39.9	BMI ≥40
All subjects	706	33.12	8.12	1.3	11.6	24.2	25.1	19.6	18.3
Age (years)**									
25–34	211	31.00 [B]	7.47	33.3	48.8	33.3	28.8	25.4	19.4
35–44	169	33.19 [A;B]	8.25	22.2	17.1	29.2	22.6	23.9	23.3
45–64	271	34.46 [A]	8.24	22.2	29.3	31.6	42.9	38.4	48.1
65–74	55	34.51 [A]	8.08	22.2	4.9	5.9	5.7	12.3	9.3
Education									
None	46	30.49 [B]	7.81	11.1	8.5	10.5	5.7	3.6	3.9
1–7 years	215	33.02 [A;B]	8.27	33.3	35.4	26.9	29.9	33.3	29.5
8–12 years	414	33.33 [A;B]	7.96	55.6	53.7	57.9	59.3	58.7	61.2
Higher (tertiary)	32	34.96 [A]	9.18	0.0	2.4	4.7	5.1	4.4	5.4
Employment status									
Employed	141	32.80	8.41	11.1	19.5	22.2	22.0	18.8	16.3
Unemployed	411	32.82	7.95	55.6	63.4	57.9	59.9	55.8	55.8
Other: homemaker	11	30.14	7.92	11.1	2.4	1.2	1.7	1.5	0.8
Pensioners	112	34.96	8.21	22.2	11.0	12.9	13.0	19.6	22.5
Disabled	26	32.34	8.42	0.0	3.7	5.3	2.8	3.6	3.1
Students	5	36.70	7.93	0.0	0.0	0.6	0.6	0.7	1.6
Housing type									
Built formal unit	144	34.41 [A]	7.33	11.1	13.4	16.4	21.5	27.5	21.7
Council/core	273	33.40 [A;B]	8.46	33.3	40.2	36.3	39.6	38.4	40.3
Informal shack	289	32.23 [B]	8.10	55.6	46.3	47.4	39.0	34.1	38.0
Urbanisation									
<20%	89	31.74	7.92	0.0	15.9	16.4	10.7	11.6	9.3
20–69.9%	340	33.17	7.79	33.3	45.1	45.6	53.7	49.3	45.7
≥70%	278	33.50	8.56	66.7	39.0	38.0	35.6	39.1	45.0
Asset index by tertiles***									
1st (poorest)	221	30.49 [C]	7.26	44.4	51.2	38.6	29.4	26.1	16.3
2nd	263	33.48 [B]	8.48	44.4	32.9	39.2	33.9	34.8	44.2
3rd (richest)	222	35.32 [A]	7.80	11.1	15.9	22.2	36.7	39.1	39.5

BMI: body mass index; WC: waist circumference, HC: hip circumference; WHR: waist hip ratio; SD: standard deviation, [A], [B], [C]: different symbols indicate significant difference between groups, Bonferroni multiple comparisons, $p < 0.05$. ** Significant relationship with BMI group, Chi square test, $p < 0.01$. *** Significant relationship with BMI group, Chi square test, $p < 0.0001$.

4.3 DIETARY INTAKE

The dietary data is presented in two ways. In Tables 4.3.1–8 the data is presented with all the original participants. In Tables 4.4.1–8 the data has been recalculated using Goldberg's method (Goldberg equation = EI divided by estimated energy requirement (EER)), in order to remove under-reporters. Under-reporters are those regarded as falling below 0.8.[146] This was done for two reasons: first, the 24HR is known to underestimate dietary intake; and second, energy intakes of the study participants were found to be very low, frequently lying well below the DRI, despite the high prevalence of overweight and obesity found among the participants. The recalculated dietary data were also used in further dietary calculations and associations. To ensure comparability of the current study with the 1990 study, which was conducted in 15–64-year-old participants[34,136] the common age categories of 25–64-years were used for this analysis. The sample, after removal of the under-reporters included 330 females and 214 males (N=544).

Macronutrient intakes of males are presented in Table 4.3.1a. Overall, males had a mean energy intake of 6516 kJ, protein intake of 50.7 g, CHO intake of 200.1 g, and a fat intake of 47.3 g. The mean energy intake was considerably less than the recommended intake of 10 609 kJ and the mean protein intake was less than the RDA of 56 g. Carbohydrates were greater than the EAR of 100 g. Mean animal protein intake was higher than plant protein intake. The mean intakes of the two youngest age groups are significantly higher when compared to the older age groups for energy, plant protein, CHO and total fat intake. There were also significant differences regarding level of education. Mean energy, protein, animal protein and fat intakes were significantly lower in the group having no education compared to those with tertiary education. There were significant differences in mean intakes of protein, animal protein and total fat between those who live in formal houses compared to those living in shacks, with the latter being lower. Mean animal protein intake was significantly higher in those who had lived in the city for longer than 70% of their life. Mean protein, animal protein and fat intakes were significantly higher in those in the upper asset group compared to the lowest group.

Macronutrient intakes of females are presented in Table 4.3.1b. Overall, the females had a mean energy intake of 5760.3 kJ, protein intake of 42.5 g, CHO intake of 186 g, and a fat intake of 44.2 g. Overall, females had significantly lower energy, protein, plant and animal protein, and CHO intakes than males. Mean energy intake of females was less than the recommended 9555.5 kJ and mean protein was less than the RDA of 46 g. Mean CHO intake was greater than the EAR of 100 g. There were significant differences regarding level of education. Mean protein, animal protein and fat intakes were significantly lower in the group having no education compared to those with tertiary education. There were significant differences in mean intakes of protein, animal protein and total fat between those who live in formal houses compared to those living in shacks, with the latter being lower. Mean plant protein intakes were significantly higher in those living in shacks. Mean protein and animal protein intakes were significantly higher in those who had lived in the city for longer than 70% of their life. Mean protein and animal protein intakes were significantly higher in those in the upper asset group compared to the lowest group, while mean plant protein and CHO intakes were significantly lower.

Overall, males had a mean SF intake of 13 g, PUFA intake of 15.4 g, cholesterol intake of 244.4 g, TS intake of 42.1 g and a fibre intake of 15.5 g (Table 4.3.2a). The mean intake of fibre was less than the AI of 28 g. The AS intake was less than the 25 g recommended by the WHO. The youngest group of males had significantly higher mean intakes of SF, MUFA and PUFA compared to the oldest groups. Similarly, those with the highest level of education had significantly higher mean intakes of SF, MUFA and PUFA. Those in formal housing had significantly higher SF and MUFA than those in informal shacks. Those who had lived in the city for longer than 70% of their life had significantly higher MUFA intakes, as did those in the upper asset tertile.

Overall, females had a mean SF intake of 12 g, PUFA intake of 14.8 g, cholesterol intake of 186.2 g, TS intake of 46.8 g and a fibre intake of 13.5 g (Table 4.3.2b). There were significant differences between males and females, with females having a significantly lower mean cholesterol intake but significantly higher mean intakes of TS, AS and fibre than males. The mean intake of fibre was less than the AI of 28 g and the AS intake was less than 25 g recommended by the WHO. Females had significantly higher mean AS values than males and significantly lower cholesterol and fibre intakes. The youngest group of females had the highest mean intake of cholesterol compared to the older groups. Those with the highest level of education had significantly higher mean intakes of SF, MUFA and cholesterol. Those who had lived in the city for longer than 70% of their life had significantly higher SF and MUFA intakes. Females in the upper asset index group had significantly higher mean SF and cholesterol intakes.

Table 4.3.3a presents data on the vitamin intakes of males. Males had a mean intake of vitamin A of 966.9 ug RE, thiamine of 0.9 mg, riboflavin of 1.2 mg, niacin of 13.9 mg, vitamin B6 of 1.1 mg, folate of 282.9 ug, vitamin B12 of 7.7 ug, and vitamin C of 54.1 mg. Mean intakes of thiamine, folate and vitamin C fell below the EAR values. The mean thiamine intake was significantly the lowest in males in the oldest age group, as was the mean niacin intake.

Table 4.3.3b presents data on the vitamin intakes of females. They had a mean intake of vitamin A of 750.7 ug RE, thiamine intake of 0.8 mg, riboflavin of 1.0 mg, niacin of 11.4 mg, vitamin B6 of 1.0 mg, folate of 237.2 ug, vitamin B12 of 3.8 ug, and vitamin C of 50.5 mg. Females had significantly lower mean thiamine, niacin, B6, folate and vitamin B12 intakes than males. Mean intakes of thiamine, vitamin B6, folate and vitamin C fell below the EAR values. Those with tertiary education had the highest mean intake of niacin and those living in a shack had the lowest intake of vitamin B12. Furthermore, mean niacin and folate intakes were also significantly the highest in those who had spent >70% in an urban setting, while mean folate intakes were significantly lowest in the highest asset group.

In Table 4.3.4a, mineral intakes of adult males are presented. Overall, mean calcium intake was 310.5 mg, iron intake was 10.5 mg, and zinc 7.5 mg. All these minerals lie below the recommended values of 1000 mg for calcium and 9.4 mg for zinc, except for iron which is at 7 mg. Mean sodium intake was 1078.3 mg. Mean intake of sodium was significantly higher in the younger two age groups. In terms of education, those with tertiary education had significantly higher mean levels of phosphorus, potassium and sodium. Mean zinc intake was significantly higher in males in the younger age group and those in the highest asset group.

Mineral intakes of adult females are presented in Table 4.3.4b. Overall, mean calcium intake was 270.1 mg, iron intake was 9.1 mg and zinc was 6.2 mg. For all minerals except iron, sodium and zinc, females had significantly lower mean intakes than males. All of the mean mineral intakes of females lie below the recommended values of 1000 mg for calcium and 6.8 mg for zinc, except for iron which is at 6 mg. Mean sodium intake was 1005.4 mg. In terms of housing type, those living in shacks had significantly lower levels of mean calcium, phosphorus, and sodium, but significantly higher levels of iron and magnesium. There were significant mean differences in phosphorus, sodium and zinc intakes of those living in the city for more than 70% of their life. This was also the case for those in the highest asset group for phosphorous and sodium intakes.

Table 4.3.5a presents data on the macronutrient distribution in relation to energy intake in males. Distribution of energy intake was the following: Protein 13.2%, fat 25.6%, CHO 53.6%, TS 11.2%, and AS 9.5%. The P/S ratio was 1.5 and SF accounted for 7.1%E, MUFA 8.2%E and PUFA 8.2%E. These all fell within the acceptable macronutrient distribution ranges. The mean %E from fat, MUFA, and PUFA intakes was significantly higher in the youngest age groups compared to the two older groups, while mean %E from CHO was significantly lower in the youngest age group. Those with a tertiary education had the highest significant mean %E from animal protein, fat, MUFA, and PUFA intakes compared to those having no education; while their mean %E from CHO was significantly lower. Mean %E from animal protein, fat, SF, MUFA, and PUFA was significantly higher in adults residing in formal houses; those who had spent more than 70% of their life in a city, and those within the highest asset group. Mean %E from CHO intake was significantly lower in those in formal housing, highest urbanisation and the highest assets category.

Table 4.3.5b presents data on macronutrients distribution in relation to energy intake in females. Distribution of energy intake was the following: protein 12.6%, fat 26.6%, CHO 55, 8%, TS 14.1% and AS was 12.3%. The P/S ratio was 1.5 and SF accounted for 7.2%E, MUFA 8.4%E, and PUFA 8.8%E. These all fell within the acceptable macronutrient distribution ranges. There were significant differences between the males and females with regard to %E from CHO and TS intake, with females having higher intakes. Females in the oldest age group had the highest mean %E from TS intake. Those with a tertiary education had significantly higher mean %E from protein, animal protein, fat, SF, and MUFA intakes. The same applied to those living in formal housing and with more than 70% of life spent in a city. However, the opposite trend was found for mean %E from CHO, with those having a tertiary education, residing in formal housing and living in a city for more than 70% of their life, who had significantly lower intakes. A similar trend was found in mean %E from total and added sugar intakes.

Table 4.3.1a: Mean (standard deviation) of the energy and macronutrient intakes of black males in Cape Town by age, education level, employment status, housing, urbanisation and asset index

	N	Energy (kcal/day)		Energy[1] (kJ)		Protein[2] (g)		Plant protein (g)		Animal protein (g)		CHO (g)		Total fat (g)	
All subjects	391	1439.6	672.79	6516.0	2929.77	50.7	28.44	22.2	10.48	27.2	24.79	201.1	87.70	47.3	36.75
Age (years)															
25–34	107	1612.1 [A]	722.84	7199.5 [A]	3152.59	52.4	28.97	23.5 [A]	9.85	26.3	24.24	212.5 [A]	94.33	58.7 [A]	36.33
35–44	105	1466.3 [A]	618.69	6687.8 [A]	2711.05	54.6	30.83	21.5 [A;B]	9.49	32.3	27.77	201.5 [A;B]	72.27	49.4 [A;B]	36.90
45–64	148	1366.0 [A;B]	676.00	6226.9 [A;B]	2937.40	48.4	26.49	22.4 [A;B]	11.83	24.7	23.19	200.6 [A;B]	94.84	41.3 [B;C]	37.03
65–74	31	1104.4 [B]	469.73	4955.3 [B]	2003.09	43.3	25.91	18.6 [B]	8.09	24.6	21.86	163.2 [B]	66.44	29.0 [C]	21.34
Education															
None	27	1217.2 [B]	920.42	5548.1 [B]	4038.80	40.2 [B]	26.48	24.3	17.18	14.9 [B]	17.27	207.6	159.10	22.3 [C]	17.84
1–7 years	128	1351.6 [B]	608.20	6173.6 [B]	2718.92	47.7 [A;B]	29.31	21.8	10.29	25.3 [A;B]	26.06	194.0	73.18	42.5 [B]	38.43
8–12 years	210	1475.5 [A;B]	658.55	6656.6 [A;B]	2837.66	52.9 [A;B]	27.83	21.6	9.45	29.5 [A]	24.37	201.5	83.76	50.5 [A;B]	35.03
Higher (tertiary)	27	1791.1 [A]	675.07	7978.2 [A]	2887.41	58.9 [A]	27.79	26.2	10.22	30.6 [A]	24.89	226.0	86.84	69.1 [A]	39.57
Employment status															
Employed	91	1548.2	605.58	7064.6	2680.15	61.3	31.04	22.3	10.58	37.3	27.15	205.8	79.75	55.2	32.90
Unemployed	235	1478.2	723.72	6647.4	3128.46	49.2	27.56	22.5	10.81	25.2	23.86	204.8	93.22	48.9	39.85
Other: homemaker	4	1750.5	697.95	8293.4	3554.04	68.1	43.59	24.7	10.60	38.3	37.09	263.3	106.38	45.2	26.06
Pensioners	40	1077.1	421.87	4928.0	1832.19	40.0	23.09	20.4	8.94	19.6	19.92	174.0	71.61	25.7	16.09
Disabled	19	1125.2	430.48	5187.2	1786.11	39.8	17.66	21.6	10.04	18.2	18.05	180.1	73.04	30.6	26.50
Students	3	1406.0	542.28	6274.4	2433.78	40.2	12.15	19.5	4.50	19.9	15.66	173.2	67.05	63.9	35.77
Housing type															
Built formal unit	87	1548.5	664.74	6914.3	2742.59	56.1 [A]	28.13	21.8	9.36	32.7 [A]	24.07	201.0	78.53	57.5 [A]	41.17
Council/core house/hostel	115	1388.0	566.27	6307.6	2410.39	53.5 [A;B]	28.28	21.1	8.35	30.5 [A]	25.79	189.9	69.20	47.1 [A;B]	32.69
Informal shack	190	1420.6	730.63	6458.7	3273.12	46.6 [B]	28.20	23.0	11.98	22.7 [B]	23.81	207.9	100.40	42.7 [B]	36.15
Urbanisation															
<20%	44	1433.6	818.63	6563.8	3663.16	49.7 [A;B]	32.83	24.8	11.13	22.2 [B]	26.26	223.1	118.27	40.3	35.98
20–69.9%	170	1367.6	645.16	6180.2	2844.77	45.2 [B]	24.31	21.7	11.58	22.6 [B]	20.30	200.2	94.31	42.6	32.01
≥70% in urban	178	1509.7	656.50	6825.2	2792.29	56.3 [A]	30.02	22.0	9.09	32.8 [A]	27.20	196.7	70.70	53.4	40.25
Asset index by tertiles															
1st (poorest)	142	1446.6 [A;B]	819.27	6563.9	3637.35	47.4 [B]	31.24	25.0 [A]	12.64	20.9 [B]	25.97	216.5 [A]	108.43	41.8 [B]	40.98
2nd	115	1327.3 [B]	592.35	6079.1	2571.73	48.2 [A;B]	23.89	19.9 [B]	7.98	27.2 [A;B]	20.69	180.6 [B]	66.61	44.2 [B]	34.05
3rd (richest)	135	1527.8 [A]	546.75	6838.2	2293.87	56.4 [A]	28.27	21.2 [B]	9.20	33.8 [A]	25.21	202.6 [A;B]	75.58	55.7 [A]	32.84

[1] Sedentary man of 1.75 m with BMI of 24.9 kg/m^2: 10 609 kJ; [2] Recommended Dietary Allowance protein: 56 g; Estimated average requirement CHO: 100 g; CHO: Carbohydrate; SD: Standard deviation; [A], [B], [C]: different symbols indicate significant difference between groups, Bonferroni multiple comparisons, $p < 0.05$; * Significant relationship with BMI group, Chi square test $p < 0.05$; ** Significant relationship with BMI group, Chi square test, $p < 0.01$; *** Significant relationship with BMI group, Chi square test, $p < 0.0001$.

Table 4.3.1b: Mean (standard deviation) of the energy and macronutrient intakes of black females in Cape Town by age, education level, employment status, housing, urbanisation and asset index

	N	Energy (kcal/day)		Energy[1] (kJ)		Protein[2] (g)		Plant protein (g)		Animal protein (g)		CHO (g)		Total fat (g)	
All subjects	706	1265.9 ***	579.08	5760.3 ***	2445.95	42.5***	22.41	19.2 ***	8.74	21.6***	19.84	186.0**	74.23	44.2	35.09
Age (years)															
25–34	211	1374.6 [A]	690.28	6162.2	2871.49	46.1	26.97	19.5	9.05	24.4	24.10	191.5	79.23	51.1 [A]	39.52
35–44	169	1306.8 [A;B]	501.85	5931.2	2173.23	42.3	20.55	19.7	8.68	21.3	17.78	194.4	67.69	45.2 [A;B]	32.23
45–64	271	1165.8 [B]	497.02	5372.1	2099.90	39.8	18.87	18.7	7.97	19.5	16.87	177.8	66.87	38.6 [B]	32.69
65–74	55	1216.6 [A;B]	634.15	5606.7	2792.28	42.0	23.30	18.5	11.13	21.6	20.56	179.6	100.97	42.2 [A;B]	32.87
Education															
None	46	1098.8	568.11	5073.1	2460.76	36.1 [B]	21.04	19.3	12.56	15.2 [B]	19.21	183.2	94.38	29.2 [B]	32.22
1–7 years	215	1153.5	461.88	5331.5	1993.76	38.7 [B]	19.04	19.2	8.58	18.5 [A;B]	16.35	181.6	69.14	36.1 [A;B]	27.45
8–12 years	414	1341.9	624.67	6058.5	2622.87	44.7 [A;B]	23.62	19.4	8.45	23.5 [A;B]	21.25	190.1	74.39	49.5 [A]	38.07
Higher (tertiary)	32	1280.2	541.11	5781.5	2288.15	49.2 [A]	24.30	15.6	6.21	27.0 [A]	19.19	167.0	71.25	51.1 [A]	28.89
Employment status															
Employed	141	1276.7	604.36	5799.3	2524.00	41.2	19.74	18.1	7.77	20.6	17.15	182.8	72.71	47.5	38.76
Unemployed	412	1280.7	575.25	5818.9	2422.13	43.7	23.92	19.6	8.79	22.5	21.16	188.8	71.69	43.9	33.63
Other: homemaker	11	1312.6	722.57	5908.1	2950.09	46.5	20.29	19.0	11.21	27.4	22.36	157.4	77.62	60.2	50.32
Pensioners	112	1198.6	600.88	5504.0	2603.50	40.4	20.86	18.5	9.66	19.8	18.26	180.4	86.57	40.6	36.79
Disabled	26	1275.3	350.96	5800.6	1482.42	40.1	19.55	22.3	7.17	17.8	18.68	202.5	61.40	37.9	20.44
Students	5	1101.8	335.20	5053.3	1899.85	33.0	13.99	17.1	7.55	15.8	10.40	151.2	71.09	46.9	17.94
Housing type															
Built formal unit	144	1211.8	612.90	5564.3	2444.44	44.5 [A]	20.32	17.3 [B]	7.56	25.0 [A]	19.14	172.8	70.42	44.4 [A;B]	38.47
Council/core house/hostel	274	1335.9	572.21	6040.6	2463.56	44.9 [A]	22.99	19.9 [A]	8.52	23.6 [A]	19.77	188.9	71.71	49.0 [A]	36.71
Informal shack	289	1226.8	563.44	5593.3	2413.41	39.2 [B]	22.51	19.4 [A]	9.37	17.9 [B]	19.73	189.9	77.83	39.5 [B]	31.02
Urbanisation															
<20%	89	1236.4	519.66	5594.4	2284.18	36.9 [B]	17.79	18.3	7.45	17.4 [B]	14.96	189.2	70.80	41.2	34.33
20–69.9%	340	1250.3	563.39	5731.6	2379.15	41.4 [A;B]	22.75	20.0	9.24	19.7 [B]	20.26	190.4	72.96	41.7	33.79
≥70% in urban	278	1294.4	615.47	5848.1	2577.44	45.6 [A]	22.88	18.5	8.42	25.2 [A]	20.17	179.6	76.58	48.1	36.63
Asset index by tertiles															
1st (poorest)	221	1229.6	561.77	5592.1	2410.75	38.0 [B]	21.02	20.0 [A]	9.77	17.0 [B]	17.97	189.7 [A]	77.28	39.5	31.86
2nd	264	1325.8	594.76	6038.0	2521.24	43.7 [A]	22.98	19.7 [A]	8.46	22.0 [A]	20.65	194.7 [A]	73.29	47.2	37.92
3rd (richest)	222	1231.1	573.93	5598.9	2370.11	45.5 [A]	22.46	17.7 [B]	7.78	25.6 [A]	19.80	172.0 [B]	70.42	45.2	34.35

[1] Sedentary woman of 1.60 m with BMI of 24.9 kg/m^2: 9555.5 kJ. [2] Recommended Dietary Allowance protein: 46 g; Estimated average requirement CHO: 100 g; CHO: Carbohydrate; SD: Standard deviation; [A], [B], [C]: different symbols indicate significant difference between groups, Bonferroni multiple comparisons, p < 0.05. * Significant difference between males and females p <0.05; ** Significant difference between males and females p <0.01; **** Significant difference between males and females p <0.05 (Independent t test).

Table 4.3.2a: Mean (standard deviation)of the daily intake of fat, cholesterol, sugar and fibre of black male participants by age, education level, employment status, housing, urbanisation and asset index

	N	SF (g)		MUFA (g)		PUFA (g)		Trans fats (g)		Cholesterol (mg)		[1]Total sugar (g)		Added sugar (g)		[2]Dietary fibre (g)	
All subjects	390	13.0	10.87	15.2	12.89	15.4	16.06	0.6	1.55	244.4	307.94	42.1	36.76	36.5	37.03	15.5	9.77
Age (years)																	
25–34	107	16.3 [A]	13.13	19.0 [A]	13.81	18.8 [A]	15.75	1.0	2.35	261.8 [A;B]	316.37	40.3	38.68	39.6	44.82	16.8	10.74
35–44	105	12.9 [A;B]	9.64	15.8 [A;B]	12.98	16.3 [A]	15.22	0.5	0.88	341.2 [A]	359.49	45.6	40.60	35.3	33.98	14.9	8.32
45–64	148	11.3 [B]	9.89	13.3 [B;C]	12.18	13.7 [A;B]	17.56	0.5	1.28	183.1 [B]	265.20	41.9	33.71	37.4	35.05	15.5	10.08
65–74	31	9.3 [B]	7.58	9.4 [C]	8.39	7.7 [B]	6.83	0.4	0.77	149.4 [B]	157.41	37.4	30.26	25.0	22.89	13.0	9.02
Education																	
None	27	6.0 [B]	4.19	6.8 [C]	6.01	7.6 [B]	8.63	0.2	0.32	112.2 [B]	237.26	36.6	34.60	30.3	32.79	15.8	11.39
1–7 years	128	11.4 [A;B]	10.74	12.9 [B;C]	12.46	15.1 [B]	18.29	0.4	0.79	215.4 [A;B]	308.34	38.4	31.14	32.6	30.91	14.8	9.95
8–12 years	210	14.2 [A]	10.83	16.8 [A;B]	12.81	15.2 [B]	14.41	0.8	1.88	284.4 [A]	321.39	45.0	39.89	38.2	39.66	15.4	9.38
Higher (tertiary)	27	17.3 [A]	12.50	22.1 [A]	14.70	24.8 [A]	18.56	1.0	1.90	198.4 [A;B]	191.99	41.6	37.28	46.7	44.50	18.6	10.17
Employment status																	
Employed	91	15.6	10.61	18.4	12.76	16.5	12.22	0.7 [A;B]	1.27	293.1	310.92	44.6	42.67	37.0	36.44	15.9	9.68
Unemployed	235	13.1	11.37	15.7	13.62	16.4	18.25	0.6 [A;B]	1.65	249.7	323.30	41.0	34.38	36.5	37.77	15.5	9.98
Other: homemaker	4	9.6	3.51	13.8	6.88	17.6	15.26	0.1 [B]	0.10	322.1	328.65	47.5	46.50	46.5	48.10	19.8	9.49
Pensioners	40	8.4	6.60	8.1	6.55	7.0	6.33	0.4 [A;B]	0.74	134.9	172.31	40.6	34.24	30.8	28.35	14.5	9.38
Disabled	19	6.9	5.62	8.4	6.60	13.1	14.73	0.6 [A;B]	2.22	139.0	233.43	47.9	42.34	38.4	39.58	14.5	9.52
Students	3	21.7	22.05	21.5	10.66	15.0	2.81	2.5 [A]	4.37	347.1	465.83	26.9	17.40	60.0	78.43	16.7	7.16
Housing type																	
Built formal unit	87	16.1 [A]	11.12	18.9 [A]	13.48	18.1	20.53	0.9	1.83	243.8	275.37	46.5	37.49	36.5	36.97	16.0	9.08
Council/core house/hostel	115	13.4 [A;B]	10.59	15.6 [A;B]	13.06	13.8	11.67	0.6	1.26	301.9	326.57	37.1	31.73	34.2	32.60	14.9	8.01
Informal shack	190	11.2 [B]	10.60	13.3 [B]	12.17	15.0	15.95	0.5	1.56	210.2	306.98	43.1	39.06	37.8	39.59	15.6	10.99
Urbanisation																	
<20%	44	11.1	12.51	12.9 [B]	13.31	13.0	11.77	0.9	2.90	229.6	303.98	49.6	45.61	44.6	54.18	18.5 [A]	11.73
20–69.9%	170	11.6	10.32	13.3 [A;B]	11.55	14.6	13.64	0.5	1.31	203.4	284.01	39.0	33.44	37.1	35.49	14.1 [B]	9.06
≥70% in urban	178	14.7	10.77	17.6 [A]	13.64	16.7	18.82	0.7	1.29	287.3	326.32	43.2	37.32	33.9	33.15	16.1 [A;B]	9.74
Asset index by tertiles																	
1st (poorest)	142	9.9 [B]	9.24	12.4 [B]	12.29	16.3	20.16	0.3 [B]	0.64	236.3	354.17	43.9 [A;B]	38.60	38.0 [A;B]	41.29	16.7	10.76
2nd	115	12.5 [B]	10.89	14.6 [B]	12.76	13.5	12.81	0.6 [A;B]	1.94	235.5	274.08	33.3 [B]	27.26	28.9 [B]	29.07	13.9	9.29
3rd (richest)	135	16.5 [A]	11.45	18.7 [A]	12.89	15.9	13.55	1.0 [A]	1.76	260.5	283.96	47.7 [A]	40.60	41.3 [A]	37.62	15.6	8.93

[1] Recommended sugar intake WHO <25 g; [2]Adequate intake dietary fibre: 28 g; SF saturated fat, MUFA monounsaturated fat; PUFA: polyunsaturated fat; SD: Standard deviation; [A], [B], [C]: different symbols indicate significant difference between groups, Bonferroni multiple comparisons, $p < 0.05$.

Table 4.3.2b: Mean (standard deviation)of the daily intake of fat, cholesterol, sugar and fibre of black female participants by age, education level, employment status, housing, urbanisation and asset index

	N	SF (g)		MUFA (g)		PUFA (g)		Trans fats (g)		Cholesterol (mg)		[1]Total sugar (g)		Added sugar (g)		Dietary fibre (g)	
All subjects	706	12.0	11.21	14.0	12.04	14.8	15.36	0.5	1.18	186.2**	242.57	46.8*	35.40	41.6*	36.63	13.5**	7.86
Age (years)																	
25–34	211	13.4	11.99	17.0 [A]	14.63	16.8	14.58	0.7	1.55	226.3 [A]	293.49	44.9	38.42	43.6	37.37	13.2	8.00
35–44	169	11.9	8.16	14.3 [A;B]	10.64	15.5	15.83	0.5	1.19	199.8 [A;B]	258.13	48.7	35.20	46.8	36.22	13.2	7.65
45–64	271	10.9	12.08	11.6 [B]	9.73	13.0	16.29	0.4	0.83	155.9 [A;B]	190.94	46.4	33.69	36.7	31.69	13.8	7.73
65–74	55	12.1	11.42	13.8 [A;B]	13.03	13.2	10.62	0.6	0.95	140.7 [B]	173.53	50.3	32.45	42.3	52.55	14.8	8.63
Education																	
None	46	7.5 [C]	7.65	8.2 [C]	9.61	11.3	16.29	0.2	0.40	91.1 [B]	144.96	48.3	36.33	38.8	34.85	13.6	8.61
1–7 years	215	10.3 [B;C]	12.68	10.8 [B;C]	8.68	12.2	11.89	0.4	0.91	124.2 [A;B]	157.21	47.4	34.67	39.7	33.76	14.2	8.77
8–12 years	414	13.0 [A;B]	10.48	16.1 [A;B]	13.25	16.5	16.97	0.6	1.36	226.3 [A]	277.83	46.9	36.31	43.4	38.45	13.2	7.37
Higher (tertiary)	32	15.9 [A]	11.10	17.2 [A]	10.87	13.6	8.52	0.7	0.89	222.7 [A]	219.78	39.8	26.57	35.5	33.43	12.6	6.34
Employment status																	
Employed	141	13.2	14.18	14.8	12.60	16.0	17.22	0.5	1.03	171.9	199.15	47.5	33.4	41.6	33.91	13.1	6.90
Unemployed	412	11.7	10.64	14.1	11.99	14.7	13.91	0.6	1.34	201.0	263.56	46.0	36.53	42.5	36.66	13.5	8.18
Other: homemaker	11	13.3	7.76	17.0	11.97	25.5	31.92	0.4	0.69	315.2	434.96	33.1	22.94	24.8	22.38	12.7	7.27
Pensioners	112	11.8	10.50	12.9	12.59	12.7	16.47	0.6	0.87	149.8	195.94	50.1	34.86	39.7	42.05	13.8	8.13
Disabled	26	10.2	5.40	11.8	7.11	13.0	10.59	0.3	0.54	153.9	172.98	51.3	35.35	42.7	30.55	16.2	7.03
Students	5	12.5	5.56	16.4	6.96	14.71	8.88	0.3	0.28	73.3	48.38	27.7	26.29	38.9	35.96	11.0	5.32
Housing type																	
Built formal unit	144	11.9 [A;B]	9.43	14.1 [A;B]	12.10	14.9	18.98	0.5	0.81	166.8 [B]	166.79	44.8	34.29	36.1	31.91	12.8	7.35
Council/core house/hostel	274	13.6 [A]	11.93	15.7 [A]	12.88	16.0	15.67	0.7	1.54	230.4 [A]	281.66	47.0	34.81	42.7	39.15	14.2	7.98
Informal shack	289	10.5 [B]	11.16	12.4 [B]	10.96	13.5	12.81	0.4	0.91	154.2 [B]	228.55	47.7	36.55	43.3	36.22	13.3	7.96
Urbanisation																	
<20%	89	10.2 [B]	8.55	12.5 [B]	10.26	15.3	18.45	0.4	0.73	177.2	241.23	53.1 [A]	43.34	47.4	38.97	12.8	8.02
20–69.9%	340	11.2 [A;B]	10.89	12.9 [A;B]	11.46	14.5	14.72	0.5	1.22	168.4	253.48	47.9 [A;B]	33.85	41.3	34.10	13.9	8.11
≥70% in urban	278	13.5 [A]	12.15	15.9 [A]	13.02	14.9	15.11	0.7	1.23	210.9	227.66	43.5 [B]	34.26	40.1	38.75	13.3	7.49
Asset index by tertiles																	
1st (poorest)	221	10.0 [B]	9.03	12.2 [B]	11.23	14.4	13.60	0.5	1.32	150.2 [B]	243.05	47.0 [A;B]	38.35	41.3 [A;B]	36.36	13.9	8.60
2nd	264	12.8 [A]	12.24	14.9 [A]	12.52	15.9	17.36	0.5	1.09	197.6 [A;B]	248.57	50.6 [A]	35.91	46.8 [A]	40.72	13.8	7.61
3rd (richest)	222	13.0 [A]	11.68	14.8 [A;B]	12.08	13.8	14.45	0.6	1.15	208.7 [A]	231.71	42.1 [B]	31.02	35.8 [B]	30.53	12.9	7.37

[1] Recommended sugar intake WHO <25 g; [2]Adequate intake dietary fibre: 28 g; SF saturated fat, MUFA monounsaturated fat; PUFA: polyunsaturated fat; SD: standard deviation; [A], [B], [C]: different symbols indicate significant difference between groups, Bonferroni multiple comparisons, *p* < 0.05; * Significant difference between males and females *p* <0.05; ** Significant difference between males and females *p* <0.01; **** Significant difference between males and females *p* <0.05 (Independent t test).

Table 4.3.3a: Mean (standard deviation) of the daily intake of vitamins of the male participants by age, education level, employment status, housing, urbanisation and asset index

	N	Vitamin A (ug RE)		Thiamine (mg)		Riboflavin (mg)		Niacin (mg)		Vitamin B6 (mg)		Folate (ug)		Vitamin B12 (ug)		Vitamin C (mg)	
All subjects	390	966.9	3525.44	0.9	0.47	1.2	2.11	13.9	8.89	1.1	0.71	282.9	188.95	7.7	35.99	54.1	116.13
Age (years)																	
25–34	107	819.3	3359.86	1.0 [A]	0.51	1.5	3.19	14.8 [A;B]	9.36	1.2 [A]	0.79	291.1	183.29	7.0	35.01	79.0	194.59
35–44	105	1371.2	4341.17	1.0 [A]	0.45	1.3	1.79	15.0 [A]	8.89	1.2 [A]	0.72	292.6	193.28	10.8	43.64	48.8	67.46
45–64	148	907.0	3344.84	0.9 [A;B]	0.46	0.9	1.36	13.0 [A;B]	8.49	1.1 [A;B]	0.67	276.7	193.23	6.8	34.27	45.4	64.40
65–74	31	390.5	694.41	0.7 [B]	0.41	0.8	0.73	10.9 [B]	8.41	0.8 [B]	0.46	251.4	176.77	4.3	9.57	27.4	44.68
Education																	
None	27	1297.0	5120.61	0.8	0.61	0.9	2.01	12.5	11.96	1.1	0.99	339.3	290.37	11.9	52.53	30.7	52.16
1–7 years	128	981.5	3291.31	0.8	0.44	1.0	1.33	12.7	8.59	1.0	0.62	284.6	193.08	8.2	34.03	34.9	44.36
8–12 years	210	930.9	3641.89	1.0	0.47	1.3	2.52	14.3	8.47	1.2	0.73	275.8	175.12	7.5	37.08	67.6	150.11
Higher (tertiary)	27	859.2	1222.61	1.0	0.49	1.3	1.66	17.2	9.47	1.3	0.61	277.0	149.28	3.3	4.08	62.6	70.5
Employment status																	
Employed	91	1343.6	4464.57	1.0	0.53	1.4	1.90	16.8	10.07	1.3	0.79	266.7	157.63	11.9	46.34	53.6	73.57
Unemployed	235	914.2	3414.79	0.9	0.47	1.2	2.39	13.5	8.46	1.2	0.72	293.6	204.63	6.8	34.19	59.5	138.12
Other: homemaker	4	241.1	106.01	1.0	0.23	0.8	0.62	18.5	11.19	1.2	0.46	282.5	112.60	1.3	1.34	147.1	194.69
Pensioners	40	346.4	631.60	0.7	0.39	0.7	0.66	10.9	7.85	0.8	0.45	267.3	181.25	3.2	8.67	27.6	46.00
Disabled	19	1323.9	3906.38	0.7	0.24	0.9	1.53	10.8	6.19	0.9	0.54	269.9	169.45	10.9	42.27	29.4	40.35
Students	3	414.5	84.00	0.6	0.20	0.7	0.42	7.2	1.68	0.6	0.17	234.0	106.17	1.2	1.78	18.3	13.03
Housing type																	
Built formal unit	87	833.7	2071.12	1.0	0.51	1.3	1.91	15.4	8.40	1.1	0.61	265.6	171.88	6.1	19.90	66.4	109.60
Council/core house/hostel	115	1018.5	3732.08	0.9	0.43	1.1	1.51	13.4	8.81	1.1	0.69	271.0	186.59	8.5	38.44	45.3	55.08
Informal shack	190	997.0	3920.73	0.9	0.48	1.2	2.48	13.4	9.13	1.2	0.77	298.2	197.42	8.0	40.11	53.7	142.94
Urbanisation																	
<20%	44	1389.8	4900.97	1.0	0.53	1.8 [A]	4.37	14.2	12.01	1.3 [A]	1.09	350.5 [A]	204.48	13.8	50.71	50.1	62.51
20–69.9%	170	768.2	2982.78	0.9	0.48	0.9 [B]	1.36	12.5	8.27	1.0 [B]	0.62	273.5 [B]	189.36	6.2	31.28	37.5	43.81
≥70% in urban	178	1053.2	3616.96	0.9	0.46	1.2 [A;B]	1.82	15.1	8.44	1.2 [A;B]	0.67	275.5 [B]	182.38	7.8	36.00	70.9	162.49
Asset index by tertiles																	
1st (poorest)	142	977.3	4007.98	0.9	0.51	1.0	1.62	13.2	9.70	1.2	0.77	325.1 [A]	206.06	8.3	40.94	39.8	48.99
2nd	115	867.9	3082.91	0.9	0.42	1.3	2.76	13.4	7.98	1.1	0.71	253.5 [B]	175.07	7.4	31.62	65.3	180.80
3rd (richest)	135	1040.3	3364.14	1.0	0.48	1.3	1.91	15.0	8.70	1.1	0.65	264.3 [B]	174.86	7.4	34.21	59.4	92.65

Estimated average requirements Vitamin A: 625 RE; Thiamine: 1.0 mg; Riboflavin: 1.1 mg; Niacin 12 mg; B6:1.1 mg; Folate: 320 ug; B12:2 ug; Vitamin C: 75 mg SD: standard deviation; [A], [B], [C]: different symbols indicate significant difference between groups, Bonferroni multiple comparisons, $p < 0.05$.

Table 4.3.3b: Mean (standard deviation) of the daily intake of vitamins of the female participants by age, education level, employment status, housing, urbanisation and asset index

	N	Vitamin A (ug RE)		Thiamine (mg)		Riboflavin (mg)		Niacin (mg)		Vitamin B6 (mg)		Folate(ug)		Vitamin B12 (ug)		Vitamin C (mg)	
All subjects	706	750.7	1682.32	0.8 **	0.41	1.0	1.44	11.4 ***	6.42	1.0***	0.52	237.2**	186.90	3.8*	14.66	50.5	64.75
Age (years)																	
25–34	211	880.1	2237.19	0.8	0.50	1.2	2.09	12.1	7.54	1.0	0.63	228.5	216.30	5.0	20.26	51.8	65.15
35–44	169	719.1	1573.14	0.8	0.38	1.0	1.45	11.3	5.99	1.0	0.48	243.1	190.36	4.0	13.41	44.5	60.27
45–64	271	666.7	1233.13	0.8	0.36	0.8	0.73	10.7	5.63	0.9	0.44	243.9	166.62	3.0	9.57	51.0	63.97
65–74	55	765.5	1442.06	0.8	0.40	0.8	0.71	11.8	6.42	0.9	0.49	219.4	147.02	3.1	13.54	61.0	78.90
Education																	
None	46	387.6	492.51	0.8	0.41	0.6	0.42	9.5 [B]	5.57	0.9	0.42	260.02	172.93	1.3	3.91	44.7	65.82
1–7 years	215	492.0	717.47	0.8	0.36	0.7	0.52	10.3 [A;B]	5.69	0.9	0.42	231.4	146.56	2.0	4.81	48.3	67.78
8–12 years	414	932.4	2104.24	0.9	0.43	1.2	1.80	12.0 [A;B]	6.68	1.0	0.55	242.4	210.19	5.1	18.68	51.3	62.67
Higher (tertiary)	32	666.1	710.48	0.8	0.45	1.1	0.70	12.8 [A]	7.38	1.1	0.67	177.1	102.31	3.1	3.72	62.4	69.94
Employment status																	
Employed	141	688.0	1057.09	0.7	0.38	0.8	0.60	10.8	5.86	0.9	0.51	209.2	183.00	2.6	4.86	66.0	82.90
Unemployed	412	800.4	1958.4	0.8	0.44	1.1	1.79	11.7	6.92	1.0	0.55	246.4	200.24	4.5	17.58	45.0	57.22
Other: homemaker	11	665.5	1029.01	0.7	0.34	0.9	0.62	10.5	4.65	0.8	0.34	201.3	128.86	4.4	8.21	53.9	62.25
Pensioners	112	711.7	1426.82	0.8	0.40	0.9	0.77	11.1	5.74	0.9	0.46	237.8	151.92	3.4	13.45	52.5	68.30
Disabled	26	597.4	1023.24	0.9	0.33	0.8	0.56	10.9	4.54	1.0	0.34	266.0	148.15	1.4	2.32	48.9	40.49
Students	5	287.3	91.24	0.7	0.22	0.5	0.37	8.80	2.82	0.6	0.21	189.7	88.52	0.9	0.73	16.0	9.79
Housing type																	
Built formal unit	144	574.3 [B]	828.69	0.8	0.44	0.9 [B]	0.67	11.8	6.04	0.9	0.47	202.8 [B]	141.14	2.8 [A;B]	4.53	51.3	64.15
Council/core house/hostel	274	973.9 [A]	2364.60	0.8	0.42	1.3 [A]	2.13	11.8	7.20	1.0	0.59	254.2 [A]	218.23	6.1 [A]	22.00	53.2	66.10
Informal shack	289	627.8 [A;B]	1108.37	0.8	0.39	0.7 [B]	0.64	10.7	5.73	0.9	0.47	238.3 [A;B]	172.56	2.2 [B]	7.13	47.4	63.83
Urbanisation																	
<20%	89	809.4	1652.80	0.8	0.41	0.7 [B]	0.70	10.3 [B]	5.57	0.9	0.44	247.2	205.76	3.7	12.09	49.0	62.13
20–69.9%	340	675.4	1308.58	0.8	0.40	0.9 [A;B]	1.52	10.6 [B]	5.96	0.9	0.49	249.50	210.12	2.6	5.73	45.1	59.99
≥70% in urban	278	824.2	2058.18	0.9	0.43	1.1 [A]	1.50	12.6 [B]	7.00	1.0	0.57	219.0	145.01	5.4	21.37	57.5	70.47
Asset index by tertiles																	
1st (poorest)	221	632.0	1391.58	0.8	0.42	0.8	1.41	10.3 [B]	6.14	0.9	0.52	261.0 [A]	215.97	2.4	6.18	48.5	70.79
2nd	264	899.7	2140.62	0.8	0.40	1.0	1.44	11.6 [A;B]	6.39	1.0	0.51	237.1 [A;B]	172.83	4.9	20.73	52.0	58.28
3rd (richest)	222	692.3	1274.55	0.8	0.43	1.1	1.46	12.1 [A]	6.59	1.0	0.53	213.8 [B]	168.75	4.0	11.61	50.6	65.96

Estimated average requirements Vitamin A: 500 RE; Thiamine:0.9 mg; Riboflavin:0.9 mg; Niacin 11 mg; B6:1.1 mg; Folate: 320 mg; B12:2 ug; Vitamin C:60 mg; SD: standard deviation; [A], [B], [C]: different symbols indicate significant difference between groups, Bonferroni multiple comparisons, $p < 0.05$; * Significant difference between males and females $p < 0.05$; ** Significant difference between males and females $p < 0.01$; **** Significant difference between males and females $p < 0.05$ (Independent t test).

Table 4.3.4a: Mean (standard deviation) of the daily intake of minerals for the male participants by age, education level, employment status, housing, urbanisation and asset index

	N	Calcium (mg)		Iron (mg)		Magnesium (mg)		Phosphorous (mg)		Potassium (mg)		Sodium (mg)		Zinc (mg)	
All subjects	391	310.5	284.13	10.5***	5.31	211.3	99.96	773.8	394.12	1579.5	751.03	1078.3	822.32	7.5***	4.19
Age (years)															
25–34	107	355.1	335.78	11.3 [A]	5.69	223.2	105.32	812.1	413.94	1698.0	827.08	1316.3 [A]	884.16	8.0 [A;B]	4.50
35–44	105	305.5	250.45	11.2 [A]	5.30	208.0	83.14	822.8	384.14	1594.9	671.28	1126.9 [A;B]	821.92	8.1 [A]	4.48
45–64	148	268.4	233.49	9.8 [A;B]	4.92	209.3	107.80	727.4	371.68	1519.4	730.82	949.6 [B;C]	773.25	7.1 [A;B]	3.71
65–74	31	374.6	382.63	8.3 [B]	4.95	191.6	94.35	697.7	441.82	1405.6	797.67	706.1 [C]	567.26	6.2 [B]	3.89
Education															
None	27	216.1	214.42	10.0	7.57	219.9	162.12	678.7 [B]	487.30	1358.4 [B]	714.60	601.5 [B]	514.58	6.4	4.52
1–7 years	128	305.1	290.30	9.6	4.82	202.9	90.47	745.1 [A;B]	386.04	1494.4 [A;B]	718.17	988.3 [A;B]	814.89	6.8	3.78
8–12 years	210	319.6	297.76	11.0	5.30	210.1	96.94	786.1 [A;B]	390.85	1621.7 [A;B]	774.27	1174.8 [A]	843.59	8.0	4.23
Higher (tertiary)	27	356.5	174.59	11.1	4.60	252.4	82.72	906.2 [A]	336.48	1867.4 [A]	668.03	1212.9 [A]	750.52	8.5	4.87
Employment status															
Employed	91	382.0	331.07	11.1	5.12	226.8	100.83	915.7	432.52	1782.0	827.80	1132.7	791.83	8.9	4.68
Unemployed	235	293.2	253.97	10.6	5.58	211.6	100.10	753.3	365.01	1575.5	715.73	1102.0	872.26	7.5	4.16
Other: homemaker	4	251.5	107.36	9.4	2.24	261.3	143.93	949.6	628.49	1818.0	913.04	954.4	398.01	6.6	1.48
Pensioners	40	292.5	344.41	8.9	4.84	188.3	100.67	646.5	415.14	1329.1	748.48	863.1	695.04	5.9	3.00
Disabled	19	235.4	266.79	9.4	3.87	173.1	77.65	591.9	279.68	1185.1	505.44	982.1	653.91	5.8	2.61
Students	3	283.8	128.91	10.3	2.04	193.1	14.76	655.0	182.77	1186.7	329.50	1141.4	370.45	5.0	1.91
Housing type															
Built formal unit	87	353.6	277.29	10.7	4.41	217.4	84.39	840.7	358.80	1720.5	735.89	1180.9	782.59	8.5	4.76
Council/core house/hostel	115	316.0	281.78	10.2	4.96	206.3	86.07	787.4	387.20	1536.4	715.51	1111.5	851.77	7.4	4.01
Informal shack	190	287.5	287.62	10.5	5.9	211.5	113.73	735.1	410.65	1540.8	774.35	1011.4	820.31	7.2	3.97
Urbanisation															
<20%	44	311.3	370.73	11.8 [A]	6.99	220.8	128.17	760.3	517.68	1557.0	893.09	1094.3	1015.58	8.0	5.60
20–69.9%	170	299.9	268.28	9.8 [B]	5.14	198.0	103.19	710.8	369.87	1457.7	659.66	980.7	791.74	6.8	3.38
≥70% in urban	178	320.5	276.30	10.8 [A;B]	4.93	221.8	87.49	837.3	374.36	1701.3	780.24	1167.6	793.40	8.1	4.41
Asset index by tertiles															
1st (poorest)	142	285.1	280.64	11.1	6.20	217.7	121.00	743.7	436.25	1556.2	844.92	1042.8	872.79	7.2 [B]	4.31
2nd	115	291.3	264.40	9.5	4.63	200.7	91.34	746.1	373.74	1491.3	653.36	1101.2	865.91	6.8 [B]	3.32
3rd (richest)	135	353.4	300.59	10.7	4.74	213.8	81.11	828.9	360.21	1679.0	717.89	1095.7	729.55	8.6 [A]	4.55

Adequate intake calcium: 1000 mg; Estimated average requirement iron: 8.1 mg; zinc: 9.4 mg; SD: standard deviation; [A], [B], [C]: different symbols indicate significant difference between groups, Bonferroni multiple comparisons, $p < 0.05$.

Table 4.3.4b: Mean (standard deviation) of the daily intake of minerals for the female participants according by age, education level, employment status, housing, urbanisation and asset index

	N	Calcium (mg)		Iron (mg)		Magnesium (mg)		Phosphorous (mg)		Potassium (mg)		Sodium mg)		Zinc (mg)	
All subjects	706	270.1*	229.76	9.1	4.29	174.2***	75.03	620.3***	302.42	1367.6***	647.89	1005.4	749.08	6.2	3.03
Age (years)															
25–34	211	271.3	239.69	9.4	4.81	173.1	78.64	655.9	352.13	1398.0	718.71	1125.8	877.32	6.5	3.65
35–44	169	263.0	220.10	9.3	4.32	175.1	73.74	618.6	287.66	1345.1	573.36	1074.8	762.60	6.4	2.91
45–64	271	271.2	237.47	8.9	3.79	173.6	69.43	593.8	266.00	1341.4	612.79	883.3	625.13	5.9	2.43
65–74	55	281.7	180.98	8.8	4.41	179.1	91.62	619.8	303.49	1449.3	745.47	931.5	645.87	6.1	3.30
Education															
None	46	243.3 [B]	277.87	8.5	4.36	171.1	99.35	542.8 [B]	306.27	1272.4	644.31	717.8 [C]	569.26	5.4	2.44
1–7 years	215	259.3 [B]	245.59	8.5	3.78	171.3	73.57	574.7 [A;B]	283.10	1316.7	651.75	856.7 [C;B]	655.70	5.6	2.45
8–12 years	414	271.3 [A;B]	213.71	9.5	4.51	176.5	73.53	647.0 [A;B]	308.44	1401.2	649.56	1097.8 [A;B]	784.37	6.6	3.29
Higher (tertiary)	32	365.6 [A]	231.82	8.9	4.05	169.0	65.65	693.5 [A]	296.15	1413.6	595.85	1224.7 [A]	840.39	6.6	2.96
Employment status															
Employed	141	285.2	218.86	8.4	3.79	169.2	72.84	607.0	277.12	1388.0	639.37	1031.4	738.06	5.9	2.68
Unemployed	412	252.0	224.18	9.4	4.59	175.0	74.73	625.3	316.92	1353.4	655.03	1025.9	791.84	6.4	3.22
Other: homemaker	11	270.6	202.40	8.6	3.61	166.7	68.26	661.5	323.44	1322.0	369.13	871.6	529.38	6.3	2.97
Pensioners	112	317.4	265.66	9.0	4.07	175.8	84.15	619.2	295.45	1389.3	686.54	917.0	671.71	6.0	2.90
Disabled	26	269.5	197.38	9.4	2.72	190.6	55.43	625.1	241.33	1487.9	523.68	961.4	533.57	6.4	2.16
Students	5	271.3	259.14	7.6	2.26	145.8	52.60	497.0	236.30	945.8	437.51	1090.4	454.61	5.5	1.86
Housing type															
Built formal unit	144	292.0 [A]	213.60	8.5 [B]	3.66	165.9 [B]	63.54	627.2 [A;B]	241.72	1364.5	575.91	944.6 [B]	667.55	6.2	2.86
Council/core house/hostel	274	292.4 [A]	257.30	9.7 [A]	4.66	183.4 [A]	76.35	664.9 [A]	337.35	1410.4	646.52	1151.6 [A]	842.96	6.5	3.10
Informal shack	289	238.0 [B]	205.65	8.9 [A;B]	4.17	169.7 [A;B]	78.33	574.8 [B]	288.79	1328.7	681.79	897.5 [B]	668.87	6.0	3.02
Urbanisation															
<20%	89	232.3	191.84	9.0	4.46	164.6	68.66	557.2 [B]	263.06	1284.0	683.15	855.1 [B]	600.14	5.7 [B]	2.69
20–69.9%	340	268.6	218.33	9.0	4.27	175.7	73.78	608.2 [A;B]	285.58	1355.3	619.84	969.0 [A;B]	747.04	6.0 [A;B]	2.93
≥70% in urban	278	283.8	252.61	9.3	4.27	175.5	78.43	655.0 [A]	329.49	1409.1	668.91	1097.5 [A]	784.03	6.6 [A]	3.20
Asset index by tertiles															
1st (poorest)	221	236.0 [B]	218.55	9.0	4.62	173.5	81.57	572.2 [B]	305.16	1315.6	711.29	894.8 [B]	703.10	5.8	3.14
2nd	264	283.9 [A;B]	236.64	9.3	4.07	178.6	74.25	641.6 [A]	297.10	1432.7	619.34	1013.3 [A;B]	704.51	6.5	3.02
3rd (richest)	222	287.6 [A]	229.72	9.0	4.21	169.7	68.95	643.0 [A]	301.72	1342.2	610.15	1106.1 [A]	828.84	6.3	2.89

Adequate intake calcium: 1000 mg; Estimated average requirement iron: 6.0 mg; zinc: 6.8 mg; [A], [B], [C]: different symbols indicate significant difference between groups, Bonferroni multiple comparisons, $p < 0.05$; * Significant difference between males and females $p < 0.05$; ** Significant difference between males and females $p < 0.01$; *** Significant difference between males and females $p < 0.05$ (Independent t test).

Table 4.3.5a: Mean energy (standard deviation) distribution of macronutrients of black males by age, education level, employment status, housing, urbanisation and asset index

	N	Protein %E		Animal Protein %		Fat %E		SF %E		MUFA %E		PUFA %E		P:S ratio		CHO %E		Total sugar %E		Added sugar %E	
All subjects	391	13.2	5.21	45.2	26.55	25.6	12.63	7.1	4.50	8.2	4.97	8.2	6.13	1.5	1.19	53.6	12.59	11.2	9.17	9.5	8.96
Age (years)																					
25–34	107	12.4	5.02	42.6	25.65	29.9 [A]	11.94	8.2	5.10	9.6 [A]	5.00	9.8 [A]	6.74	1.6	1.24	50.9 [B]	11.85	9.3	7.60	8.9	8.39
35–44	105	13.5	5.32	49.5	26.45	25.5 [A;B]	11.83	6.7	3.54	8.0 [A;B]	4.52	8.4 [A;B]	5.66	1.5	1.01	52.7 [A;B]	13.12	11.6	10.13	9.0	9.23
45–64	148	13.5	5.42	43.7	27.15	23.6 [B]	13.01	6.6	4.59	7.7 [A;B]	5.14	7.5 [A;B]	6.00	1.6	1.26	55.4 [A;B]	12.40	11.8	9.42	10.1	9.05
65–74	31	14.0	4.35	46.5	26.46	20.5 [B]	11.97	6.5	4.35	6.5 [B]	4.71	5.7 [B]	4.84	1.3	1.19	57.2 [A]	12.44	13.2	8.95	10.0	9.74
Education																					
None	27	13.0	5.00	30.3 [B]	29.00	15.4 [C]	8.58	4.3 [B]	2.67	4.7 [B]	2.98	5.1 [B]	4.80	1.7	1.56	63.4 [A]	9.76	11.8	9.33	9.4	8.90
1–7 years	128	12.9	5.21	42.7 [A;B]	27.13	23.9 [B]	12.56	6.5 [A;B]	4.72	7.3 [A;B]	4.73	8.4 [A;B]	6.27	1.7	1.24	54.7 [B]	12.17	10.7	8.13	9.3	8.95
8–12 years	210	13.1	5.21	48.5 [A]	25.54	27.1 [A;B]	12.36	7.7 [A]	4.47	9.0 [A]	5.07	8.1 [A;B]	5.84	1.4	1.08	52.4 [B]	12.70	11.7	9.91	9.6	9.13
Higher (tertiary)	27	12.9	5.58	45.7 [A]	24.25	31.0 [A]	12.46	7.7 [A]	3.90	9.8 [A]	4.69	11.3 [A]	7.42	1.7	1.17	48.7 [B]	11.20	8.8	7.39	9.7	8.23
Employment status																					
Employed	91	14.5	4.90	55.2	22.60	28.4	11.41	8.0 [A;B]	4.31	9.4	4.88	8.5	5.05	1.4	1.01	50.0	11.89	10.6	9.72	8.9	9.05
Unemployed	235	12.7	5.34	42.7	26.84	25.8	12.79	7.0 [A;B]	4.49	8.2	4.98	8.5	6.50	1.6	1.23	53.7	12.47	10.8	8.74	9.2	8.64
Other: homemaker	4	13.1	3.77	49.4	20.36	22.6	15.01	4.7 [B]	2.01	6.9	4.37	9.0	8.34	1.6	0.85	54.1	11.68	10.9	13.14	10.9	13.59
Pensioners	40	13.4	4.88	39.4	27.63	19.5	11.64	6.3 [A;B]	5.03	6.2	5.04	5.3	4.44	1.3	1.17	59.8	12.12	14.0	9.69	11.4	9.84
Disabled	19	13.5	5.72	38.8	29.39	20.9	13.36	4.7 [B]	2.79	5.9	3.59	8.7	7.95	2.0	1.41	58.3	13.68	13.6	9.73	10.8	9.57
Students	3	11.2	3.73	43.2	30.98	36.8	7.48	11.1 [A]	7.35	12.6	1.25	9.8	3.68	1.2	0.77	46.3	6.48	7.5	5.89	12.6	13.11
Housing type																					
Built formal unit	87	13.7 [A;B]	5.02	51.9 [A]	23.10	29.3 [A]	13.30	8.5 [A]	4.93	9.7 [A]	5.17	8.8	6.95	1.3	1.08	50.3 [B]	12.43	11.8	9.32	9.3	9.03
Council/core house/hostel	115	14.2 [A]	4.87	49.6 [A]	23.94	26.5 [A;B]	12.31	7.5 [A;B]	4.49	8.7 [A]	5.27	7.8	5.13	1.4	1.08	52.0 [B]	11.73	10.1	8.72	8.9	8.36
Informal shack	190	12.4 [B]	5.38	39.4 [B]	28.35	23.3 [B]	12.07	6.2 [B]	4.11	7.2 [B]	4.47	8.1	6.29	1.7	1.27	56.0 [A]	12.72	11.5	9.36	9.9	9.30
Urbanisation																					
<20%	44	12.8	5.44	34.1 [B]	29.01	20.8 [B]	11.60	5.5 [B]	3.78	6.5 [B]	4.27	7.2	5.69	1.7	1.30	59.0 [A]	12.47	13.7	11.91	11.3	12.01
20–69.9%	170	12.5	4.74	42.5 [A;B]	26.66	24.7 [A;B]	12.35	6.7 [A;B]	4.48	7.6 [A;B]	4.70	8.5	6.40	1.6	1.27	55.7 [A]	12.12	10.9	8.07	10.1	8.79
≥70% in urban	178	14.0	5.50	50.4 [A]	24.73	27.6 [A]	12.78	7.8 [A]	4.57	9.2 [A]	5.19	8.2	5.97	1.4	1.05	50.2 [B]	12.21	10.9	9.37	8.4	8.17
Asset Index by tertiles																					
1st (poorest)	142	12.3 [B]	5.14	33.6 [B]	27.70	21.9 [B]	11.86	5.4 [C]	3.22	6.5 [C]	4.09	8.3	6.69	1.9 [A	1.32	58.0 [A]	11.91	11.5	8.78	9.5	8.97
2nd	115	13.6 [A;B]	5.13	49.1 [A]	24.44	25.4 [B]	12.82	7.2 [B]	4.57	8.3 [B]	5.08	7.8	5.78	1.4 [B	1.09	52.0 [B]	12.34	9.9	8.31	8.6	8.41
3rd (richest)	135	13.9 [A]	5.25	53.9 [A]	22.62	29.6 [A]	12.12	8.7 [A]	4.96	9.9 [A]	5.13	8.5	5.82	1.3 [B	1.03	50.3 [B]	12.26	11.9	10.17	10.2	9.41

Mean and standard deviation of energy distribution for the male participants according to background characteristics

CHO: Carbohydrate; E: energy; SF: Saturated fat, P:S ratio: Polyunsaturated fat/ saturated fat; MUFA monounsaturated fat; PUFA: polyunsaturated fat; SD: standard deviation; Acceptable macronutrient distribution range: fat: 20-35%E; CHO: 45-65%E; Protein: 10-35%E; Recommended sugar intake WHO <10%E; [A], [B], [C]: different symbols indicate significant difference between groups, Bonferroni multiple comparisons, $p < 0.05$.

Table 4.3.5b: Mean energy (standard deviation) distribution of macronutrients of black females by age, education level, employment status, housing, urbanisation and asset index

	N	Protein %E		Animal Protein %E		Fat %E		SF %E		MUFA %E		PUFA %E		P:S ratio		CHO %E		Total sugar %E		Added sugar %E	
All subjects	706	12.6	5.04	43.2	26.40	26.6	12.53	7.2	4.62	8.4	4.70	8.8	6.23	1.5	1.13	55.8**	12.33	14.1***	9.86	12.3***	9.83
Age (years)																					
25–34	211	12.8	5.24	44.5	27.28	28.5	12.00	7.3	3.81	9.3 [A]	4.74	9.6	5.79	1.6	1.05	54.1	12.02	12.6 [B]	9.32	11.9	8.88
35–44	169	12.1	4.72	43.5	25.58	26.9	11.61	7.2	3.50	8.5 [A;B]	4.31	9.0	6.25	1.5	1.11	56.3	11.78	14.1 [A;B]	8.89	13.7	10.20
45–64	271	12.8	5.04	42.2	26.17	24.9	13.11	7.1	5.67	7.6 [B]	4.69	8.2	6.54	1.5	1.19	56.9	12.50	14.8 [A;B]	10.21	11.7	9.86
65–74	55	12.6	5.22	42.5	27.02	26.5	13.5	7.4	4.88	8.5 [A;B]	5.16	8.6	6.09	1.5	1.18	55.2	13.81	16.9 [A]	12.07	12.9	11.65
Education																					
None	46	11.9 [B]	5.03	31.5 [B]	29.78	20.2 [C]	14.00	5.3 [C]	3.76	5.8 [C]	4.56	7.7	7.28	1.8 [A]	1.34	61.9 [A]	13.79	16.3	11.54	13.6	12.96
1–7 years	215	12.4 [A;B]	4.96	40.7 [A;B]	26.27	23.9 [B;C]	12.37	6.8 [C;B]	6.00	7.3 [B;C]	4.45	8.0	5.92	1.6 [A]	1.21	58.1 [A;B]	11.88	15.3	10.61	12.7	10.15
8–12 years	414	12.7 [A;B]	5.02	45.0 [A]	25.81	28.2 [A;B]	12.14	7.5 [B]	3.81	9.1 [B;A]	4.65	9.3	6.29	1.5 [A;B]	1.08	54.5 [B]	12.0	13.4	9.29	12.2	9.38
Higher (tertiary)	32	14.5 [A]	5.51	52.3 [A]	23.82	32.4 [A]	9.85	9.7 [A]	3.21	10.8 [A]	3.98	9.2	5.40	1.0 [B]	0.7	48.7 [C]	11.26	12.3	8.1	9.7	7.76
Employment status																					
Employed	141	12.4	4.91	43.8	25.39	29.0	12.43	7.6	4.72	8.8 [A;B]	4.43	9.4	6.66	1.5	1.02	54.6 [A;B]	11.37	14.0	8. 73	11.9	8.26
Unemployed	412	12.7	5.11	43.6	26.89	26.2	12.21	7.0	4.71	8.3 [A;B]	4.61	8.8	5.99	1.6	1.14	56.1 [A;B]	12.17	13.7	9.95	12.6	10.25
Other: homemaker	11	14.7	7.23	53.2	29.84	35.1	14.08	8.5	3.45	10.3 [A;B]	4.22	13.5	8.45	1.8	1.39	45.9 [B]	14.91	10.4	7.05	7.7	6.01
Pensioners	112	12.5	4.88	41.7	25.93	25.4	13.56	7.5	4.59	8.0 [B]	5.26	7.8	6.27	1.4	1.17	56.8 [A;B]	13.48	16.2	11.08	12.3	10.50
Disabled	26	11.5	4.52	35.8	24.92	24.1	11.22	6.4	3.16	7.6 [B]	4.54	8.2	5.74	1.6	1.29	58.8 [A]	12.01	14.9	9.19	12.4	8.71
Students	5	10.8	1.65	45.1	25.29	35.5	10.37	9.4	3.57	12.8 [A]	6.28	10.7	4.65	1.3	0.69	49.3 [A;B]	10.16	8.1	4.92	12.6	12.49
Housing type																					
Built formal unit	144	14.1 [A]	5.29	50.7 [A]	24.75	27.2 [A;B]	13.00	7.5 [A;B]	4.05	8.7 [A;B]	4.76	8.8	6.38	1.4 [B]	0.97	53.8 [B]	12.79	14.1	10.0	10.7 [B]	8.37
Council/core house/hostel	274	12.6 [B]	4.90	45.5 [A]	25.05	28.4 [A]	12.25	7.8 [A]	4.43	9.0 [A]	4.84	9.2	6.10	1.5 [A;B]	1.08	54.0 [B]	11.68	13.6	9.12	2.2 [A;B]	9.53
Informal shack	289	11.9 [B]	4.88	37.3 [B]	27.22	24.5 [B]	12.30	6.5 [B]	4.97	7.7 [B]	4.44	8.5	6.29	1.7 [A]	1.23	58.5 [A]	12.24	14.6	10.46	13.3 [A]	10.67
Urbanisation																					
<20%	89	11.2 [B]	3.88	39.1 [B]	26.91	25.1 [B]	12.98	6.2 [B]	3.46	7.5 [B]	3.95	9.4	7.95	1.8 [A]	1.27	58.7 [A]	12.1	15.6 [A]	10.2	14.9 [A]	11.12
20–69.9%	340	12.3 [A;B]	4.91	39.5 [B]	26.33	25.0 [B]	12.39	6.7 [B]	4.27	7.7 [B]	4.50	8.6	6.15	1.6 [A;B]	1.17	57.5 [A]	11.91	14.7 [A;B]	9.73	12.2 [A;B]	9.11
≥70% in urban	278	13.5 [A]	5.37	48.9 [A]	25.36	28.9 [A]	12.25	8.1 [A]	5.19	9.5 [A]	4.96	8.9	5.70	1.4 [B]	1.01	52.8 [B]	12.35	12.9 [B]	9.81	11.7 [B]	10.14
Asset Index by tertiles																					
1st (poorest)	221	11.4 [B]	4.43	35.8 [C]	27.63	24.6 [B]	12.95	6.1 [B]	3.87	7.5 [B]	4.64	9.1	6.68	1.8 [A]	1.25	58.6 [A]	12.13	14.4	10.8	12.8 [A;B]	10.79
2nd	264	12.4 [B]	5.12	43.3 [B]	25.83	27.0 [A;B]	11.93	7.3 [A]	4.31	8.5 [A]	4.50	9.0	6.29	1.5 [B]	1.14	55.8 [B]	11.91	14.6	9.42	13.2 [A]	9.89
3rd (richest)	222	14.0 [A]	5.20	50.4 [A]	23.80	28.0 [A]	12.62	8.1 [A]	5.42	9.2 [A]	4.86	8.4	5.68	1.3 [C]	0.89	53.1 [B]	12.46	13.3	9.35	10.9 [B]	8.55

Mean and standard deviation of energy distribution for the female participants according to background characteristics

CHO: Carbohydrate; E: energy; SF: Saturated fat, P:S ratio: Polyunsaturated fat/ saturated fat; MUFA monounsaturated fat; PUFA: polyunsaturated fat; SD: standard deviation; Acceptable macronutrient distribution range: fat: 20-35%E; CHO: 45-65%E; Protein: 10-35%E; Recommended sugar intake WHO <10%E; [A], [B], [C]: different symbols indicate significant difference between groups, Bonferroni multiple comparisons, $p < 0.05$.

Table 4.3.6 indicates the meal pattern of the participants. Approximately half of the respondents had three meals per day (55% for males and 58% for females) and more than 90% had supper (95% for males and 96% for females). Approximately 90% of the respondents had breakfast on the day of recall.

The food intake of males and females expressed as the mean and median number of portions consumed from the five basic food groups, is presented in Table 4.3.7 Milk intake was very low as less than half the recommended two portions of milk per day was consumed. Mean intake of meat was adequate for both males and females (3.2 and 2.5 portions, respectively). However, the fruit and vegetable intake was very low and did not even reach a half portion per day for both sexes (0.29 and 0.31 portions for males and females, respectively).

Table 4.3.8 shows the contribution of the different food groups to overall energy, fat, SF, cholesterol, and protein intake. The largest contribution to energy comes from the cereal group, followed by the meat group. The largest contributor to protein intake is from the meat group, followed by the cereal group, while the fat group contributes the most to total fat intake. The meat group contributes the most to SF and TC intake.

Table 4.3.6: Meal frequency and specific meals taken on the day of recall of all black participants in the Cape peninsula by gender

Number of respondents		Males (n=398)	Females (n=716)
Number of Meals	1	1.3%	2.3%
	2	18.2%	21.9%
	3	54.6%	57.5%
	4	26.0%	18.3%
Meals	Breakfast	88.9%	90.2%
	Lunch	74.1%	78.8%
	Supper	94.5%	96.4%

Table 4.3.7: Number of portions of the five basic food groups and the percentage consumers of each food group in the sample of black participants by gender

	Males (n=398)			**Females (n=716)**			**Consumers (n=1114)**
	Mean	**SD**	**Median**	**Mean**	**SD**	**Median**	
Milk Group	0.38	0.73	0.00	0.33	0.59	0.00	46.1
Milk	0.36	0.72	0.00	0.31	0.55	0.00	45.3
Cheese	0.02	0.14	0.00	0.02	0.19	0.00	2.3
Meat Group	3.2	2.93	2.64	2.5	2.18	2.11	84.0
Red meat	1.19	2.31	0.00	0.71	1.52	0.00	31.1
White meat	1.14	1.74	0.00	1.17	1.66	0.00	48.0
Eggs	0.39	0.85	0.00	0.27	0.65	0.00	17.9
Legumes	0.49	1.04	0.00	0.35	0.77	0.00	24.9
Vegetables and fruit group	0.29	0.33	0.21	0.31	0.32	0.23	77.2
Vitamin C rich vegetables/fruit	0.03	0.10	0.00	0.02	0.08	0.00	11.8
Carotene rich vegetables/fruit	0.03	0.10	0.00	0.05	0.11	0.00	27.2
Potato/sweet potato	0.15	0.28	0.00	0.14	0.24	0.00	40.3
Other veg/fruit	0.08	0.13	0.00	0.10	0.13	0.05	54.7
Cereal group	1.75	1.07	1.64	1.54	0.77	1.47	98.6
Unrefined cereals	1.07	0.98	0.86	0.88	0.70	0.86	81.9
Refined cereals	0.68	0.91	0.40	0.65	0.71	0.40	65.8
Fat group	0.36	1.26	0.00	0.40	1.29	0.00	39.1
Dripping, 'saturated fat'	0.35	1.26	0.00	0.39	1.29	0.00	10.1
Brick margarine	0.01	0.04	0.00	0.00	0.01	0.00	22.7
Oil, tub margarine	0.00	0.01	0.00	0.00	0.01	0.00	20.8

Table 4.3.8: Contribution of the five food groups to total energy, fat, cholesterol and protein intake for black males and females

	Percentage Contribution										
Food Groups		Energy		Total fat		Saturated fat		Cholesterol		Total protein	
		Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
Milk	Mean	4.2	4.2	9.9	9.2	16.4	15.8	12.7	10.9	6.5	6.5
	SD	8.1	6.9	19.1	17.1	26.2	24.4	28.7	24.8	12.4	11.9
Meat	Mean	24.5	21.8	41.2	36.4	43.5	38.4	64.7	63.9	48.7	46.9
	SD	20.0	16.3	33.0	29.2	34.1	30.8	43.1	41.9	28.6	25.8
Fruit/ vegetables	Mean	11.2	12.9	11.2	12.7	8.4	9.0	0.6	0.6	5.8	6.9
	SD	13.2	12.0	22.2	20.6	18.7	16.8	7.2	5.4	8.0	8.2
Cereal	Mean	35.6	36.3	12.6	12.0	9.8	9.3	4.1	3.8	33.0	34.4
	SD	18.1	15.8	17.0	15.1	15.8	13.9	16.9	16.6	21.4	20.0
Fat	Mean	7.4	8.9	19.7	23.8	15.2	18.7	4.0	5.9	1.6	2.1
	SD	11.3	12.1	27.2	28.3	22.9	24.9	16.0	19.2	6.8	7.7
Total %		83.0	84.1	94.6	94.0	93.3	91.3	86.1	85.1	95.6	96.7

4.4 DIETARY INTAKE WITH UNDER-REPORTERS REMOVED

In the CRIBSA study, dietary data were collected from 1097 participants of whom 1009 were between ages 25 and 64 years. Under-reporters were removed as indicated in the methods section resulting in a sample of 544 (214 males and 330 females) participants aged 25–64 years. These participants came from Khayelitsha (42.4%), Langa (31.4%), Gugulethu (15.3%), and less than 10% from Crossroads and Nyanga. Most of them had spent most of their lives in the city, the breakdown percentages are as follows: 12% spent less than 20% of their lives in the city, 44.5% spent between 20% and 69% of their lives in the city, and 43.3% spent 70–100% of their lives in the city. In terms of wealth distribution in our study sample, the poorest participants amounted to 30%, the poor 54.8% and the least poor was 14.8%.

Table 4.4.1 shows the comparison between the mean intakes for energy and macronutrients in the 2009 sample compared to the BRISK study (1990). The mean energy intakes for the males were low compared to recommended values in 1990 as well as in 2009. In females, however, the mean energy intakes were higher but still within the recommended values. Also, the mean energy intakes were significantly higher in the females in the 45–64-year age group in 2009 compared to 1990 (p <0.0001). The mean protein intakes were above the RDA for males and females in 1990 as well as in 2009, with almost half coming from plant sources. Of significance is that the mean protein intakes were lower in the males (2009=64.5 g, 1990=77.0 g) and the females (2009=52.8 g; 1990=56.0 g) when comparing 1990 to 2009 (p <0.01). This is also the case for the protein %E (p <0.01).

The mean fat intake for males and females in the younger age group (25–44 years) was significantly higher in 2009 compared to 1990 (p <0.01). In males, this was 70.3 g in 2009 compared to 60 g in 1990, while in females, this was 66.4 g in 2009 compared to 49 g in 1990. Fat %E was also found to be higher in males as well as females when comparing 2009 to 1990 (p <0.01), even though still within the recommended range. Saturated fat intake was significantly lower in 2009 (14.5 g) compared to 1990 (21 g) in the 45–64-year-old males (p <0.01). Contrary to this, in females, the SF intake was significantly higher in 2009 compared to 1990 (p <0.0001) in both age groups. Saturated fat %E was above the recommended value (<7%) for males and females in both study samples. Monounsaturated fatty acids intake decreased from 1990 to 2009 in the older males, but significantly increased in females in both age groups (p <0.01). Similarly, the PUFA intake as well as the PUFA %E was significantly higher in males and females in 2009 compared to 1990 (p <0.0001). This was also found in the diet P:S ratio. Cholesterol intake was within the <300 mg for females in 1990 as well as in 2009, however, in younger males (359.8 mg) and females (285.9 mg), it was significantly higher in 2009 compared to 1990 (M=265 mg; F=213 mg) (p <0.01).

Mean CHO intakes were more than double the EAR values for males and females in most of the age groups in both study samples. However, it was found to be significantly lower in younger males in 2009 (247.8 g) compared to 1990 (282.0 g) (p <0.01). In the females, the opposite was found. In younger females, the CHO intakes were significantly higher in 2009 (232.4 g) compared to 1990 (214.0 g) (p <0.01) and in older females, the same was found (1990=198.0 g; 2009=221.4 g) (p <0.01). The mean CHO %E was lower in 2009 than in 1990 for both males and females, but still fell within the AMDR of 45–65%. The mean fibre intakes were found to be lower than the AI of 25 g for males and females in both studies, with the older females having a significantly lower intake (13.0 g) in 1990 compared to 2009 (16.8 g) (p <0.0001). The mean AS %E intake was greater than the WHO recommendation of less than 10%, except for the younger males in 2009. Also, in females, AS intake increased significantly in the younger and older age group from 1990 (25–44 years=47.0 g; 45–64 years=38.0 g) to 2009 (25–44 years=54.4 g; 45–64 years=47.0 g) (p <0.01).

A comparison between the mean micronutrient intakes for 2009 (CRIBSA) and the BRISK study (1990) is shown in Table 4.4.2. The mean calcium intakes were far below the AI of 1000 mg for males and females in both study samples. The values for the males in both age categories were significantly higher in 1990 compared to 2009 (p <0.01). Mean iron intakes were adequate in both studies (above the EAR of 6.0 mg). Zinc and vitamin B12 intakes were also adequate in both studies (above 6.8 mg and 2.0 ug, respectively). Vitamin A intakes were also adequate (above the EAR of 500 RE), however, older males in the 1990 group had a low mean intake of 395 RE. There was also a significantly higher vitamin A intake in the females between 1990 (558 RE) and 2009 (1066 RE) (p <0.01). Thiamine intake fell within the EAR of 0.9 mg for males and females in both studies, with the exception of the females in the 1990 study. This was also true for the riboflavin intakes. Niacin intakes were also adequate (EAR for males=12 mg and females=11 mg), with the exception of the older females in the 1990 (10.0 mg) study. Vitamin B6, folate and vitamin C were below the EAR in females and males in the 1990 study. There were significant higher mean intakes for iron, folate, vitamin B6, niacin, thiamine, riboflavin and vitamin A in the 2009 study compared to 1990 (p <0.0001). The 2009 study showed adequate intakes of vitamin E (12 mg), however, for vitamin D intake, only the younger males had an adequate mean intake (5.5 ug).

Table 4.4.1: Mean (standard deviation) energy and macronutrient intakes of 25–64-year-old black adults in the CRIBSA study (2009) in Cape Town and comparison with 25–64-year-old black adults in the BRISK 1990 study[34]

	Males 2009		Males 1990		DRI[1]	Females 2009		Females 1990		DRI[1]
Age groups	25–44	45–64	25–44	45–64		25–44	45–64	25–44	45–64	
N	138	76	285	98		216	114	364	117	
Energy (kJ)	8557 ± 2971	7666 ± 2219	8500 ± 3700	9196 ± 3800	10 609 kJ[2]	7619 ± 2271**	7104± 1838***	6400± 2800	6400±2000	37 971 KJ
Protein (g)	64.5 ± 29.0**	57.0 ± 27.4**	77 ± 44.0	78 ± 51.0	RDA=56 g[4]	52.8 ± 23.4	48.7 ± 18.0	56 ± 33.0	49 ± 21.0	RDA=46 g[4]
Protein %E[5]	13.7 ± 4.8*	13.4 ± 5.1**	15.1 ±4.8	15.3 ±5.4	10–35%E[5]	12.4 ± 4.5**	12.7 ± 4.9*	14.5 ± 4.8	14.3 ± 3.3	10–35%E[5]
Plant Protein (g)	26.9 ± 11.0***	25.6 ± 10.6*	34 ± 21.0	31 ± 18.0		23.6 ± 8.9	22.9 ± 8.2	23 ± 14.0	22 ± 10.0	
Animal Protein (g)	35.5 ± 26.5*	29.1 ± 25.8**	42 ± 40.0	46 ± 49.0		26.4 ± 21.3*	23.4 ± 17.7	33 ± 30.0	28 ± 19.0	
Fat (g)	70.3 ± 41.2*	52.9 ± 35.0	60 ± 43.0	57 ± 43.0		66.4 ± 38.4***	59.9 ± 38.9***	49 ± 33.0	42 ± 25.0	
Fat %E	32.0 ± 12.1***	27.2 ± 14.0	25.9 ± 11.8	23.8 ± 11.7	20–35%E[5]	33.4 ± 11.8***	32.6 ± 14.1***	27 ± 11.2	26.1 ± 9.6	20–35%E[5]
Saturated fat (g)	18.6 ± 12.5	14.5 ± 10.3*	20 ± 17.0	21 ± 18.0		17.3 ± 11.5*	17.3 ± 16.2*	16 ± 12.0	15 ± 10.0	
Saturated fat %E[5]	8.5 ± 4.3	7.5 ± 4.5	8.6 ±4.4	8.8 ± 4.8	<7%E[5]	8.6 ± 3.8*	9.6 ± 7.9	8.8 ± 4.3	9.2 ± 3.9	<7%E[5]
MUFA6 (g)	22.6 ± 14.6	16.9 ± 12.8	21 ± 17.0	20 ± 18.0		21.7 ± 14.1**	17.7 ± 11.7	17 ± 14.0	15 ± 11.0	
MUFA %E	10.2 ± 4.8**	8.6 ± 5.5	9 ± 5.2	8.3 ± 5.0		10.8 ± 4.7***	9.8 ± 5.2	9.4 ± 5.0	9.3 ± 4.0	
PUFA7 (g)	23.5 ±19.8***	17.3 ± 16.4**	13 ± 10.0	10 ± 9.0		22.6 ± 17.1***	20.6 ± 21.7***	11 ± 9.0	8 ± 5.0	
PUFA %E[5]	10.7 ± 6.8***	8.8 ± 6.9***	5.7 ± 3.7	4.5 ± 3.4		11.5 ± 6.7***	10.8 ± 7.8***	6.3 ± 3.9	5.4 ± 3.0	
Diet P/S ratio	1.53 ± 1.1***	1.52 ± 1.3***	0.81 ± 0.67	0.73 ± 0.60		1.66 ± 1.13***	1.56 ± 1.33***	0.88 ± 0.75	0.77 ± 0.58	
Cholesterol (mg)	359.8 ± 346**	258.5 ± 322	265 ± 288	260 ± 270	300 mg	285.9± 326***	216.3 ± 227	213 ±226	174± ±136	300 mg
Carbohydrate (g)	247.8 ± 97.9**	237.5 ± 67.4	282 ± 128	266 ± 112	EAR=100 g[8] RDA=130 g[4]	232.4 ± 66.4*	221.4 ± 65.9*	214 ± 95.0	198 ± 73.0	EAR=100 g[8] RDA=130 g[4]
Carbohydrate %E[5]	53.2 ± 13.7	57.4 ± 14.1	61.3 ±16.3	59.2 ± 16.6	45–65%E[5]	55.5 ± 12.5	57.3 ± 15.0	62 ± 15.3	62.7 ± 12.2	45–65%E
Fibre (g)	18.9 ± 10.4	18.1 ± 10.4	21 ± 15.0	19 ± 13.0	AI=38 g[9]	16.2 ± 8.5	16.8 ± 8.2***	16 ± 11.0	13 ± 6.0	AI=25 g[9]
Added Sugar (g)	45.0 ± 42.8	49.4 ± 37.7	50 ± 44.0	51 ± 39.0		54.4 ± 40.8*	47.0 ± 36.3*	47 ± 34.0	38 ± 24.0	
Sugar %E[5]	9.5 ± 8.3	12.1 ± 9.3	11.0±9.6	11.4±10.1	<10%E[5]	13.3 ± 9.6	12.2 ± 8.9	13.6±11.1	11.4±6.6	<10%E[5]

[1] Dietary reference intakes; [2] Sedentary man of 1.75 m with BMI of 24.9 kg/m^2; [3] Sedentary woman of 1.60 m with BMI of 24.9 kg/m^2; [4] RDA: Recommended dietary allowance; [5] Energy; [6] MUFA=monounsaturated fatty acids; [7] PUFA=polyunsaturated fatty acids; [8] EAR: Estimated average requirement; [9] AI: Adequate intake; SD: standard deviation; * p <0.05; ** p <0.01; *** p <0.0001 using the t-test between same age categories between the two studies.

Table 4.4.2: Mean (standard deviation) mineral and vitamin intakes of 25–64-year-old black adults in the CRIBSA study (2009) in Cape Town and comparison with 25–64-year-old black adults in the BRISK 1990 study[34]

	Males 2009		Males 1990			Females 2009		Females 1990		
Age groups	25–44	45–64	25–44	45–64	DRI[1]	25–44	45–64	25–44	45–64	DRI[1]
N	138	76	285	98		216	114	364	117	
Calcium (mg)	384 ± 307***	324±245**	526±484	522±409	AI[2]=1000	312 ± 266	346 ± 275	337±221	358±208	AI=1000 mg
Iron (mg)[5]	13.3 ± 5.5***	11.4 ± 4.8	10±6	10±6	EAR[3]=8.1	11.3 ± 4.8***	10.9 ± 3.7***	7±4	7±4	EAR=6.0 mg
Zinc (mg)[5]	9.6 ± 4.5	8.5 ± 3.9*	10.6±8.4	11.8±13.4	EAR=9.4	7.8 ± 3.4	7.4 ± 2.4	7.7±6.2	6.8±4.1	EAR=6.8 mg
Vitamin A (ug)	1133 ± 3667	1198 ± 243*	724±2829	395±691	EAR=625	1066 ± 2411**	785 ± 1332	558±1141	577±1075	EAR=500 RE
Thiamin (mg)[5]	± 0.5*	1.0 ± 0.4	1.1±0.6	1.0±0.6	EAR=1.0	1.0 ± 0.5**	1.0 ± 0.4***	0.9±0.5	0.8±0.3	EAR=0.9 mg
Riboflavin (mg)[5]	1.7 ± 2.9*	1.1 ± 1.6	1.2±1.3	1.1±0.7	EAR=1.1	1.5 ± 2.3***	1.1 ± 0.8***	0.8±0.6	0.8±0.5	EAR=0.9 mg
Niacin (mg)[5]	18.1 ± 8.9*	16.0 ± 9.4	16.1±11.6	15.1±8.9	EAR=12	14.1 ± 7.4**	13.2 ± 6.2***	12.2±8	10±5.7	EAR=11 mg
Vitamin B6 (mg)[5]	1.5 ± 0.7***	1.3 ± 0.7*	1.2±0.8	0.8±0.1	EAR=1.1	1.0 ± 0.6*	0.8 ± 0.4**	0.9±0.6	0.8±0.4	EAR=1.1 mg
Folate (ug)[5]	335 ± 191***	324 ± 201***	218±175	182±147	EAR=320	289 ± 254***	302 ± 173***	155±131	147±79	EAR=320 ug
Vitamin B12 (ug)	9.8 ± 38.2	9.6 ± 43.0	6.9±29.3	3.8±5.0	EAR=2.0	5.8 ± 21.1	4.0 ± 10.6	3.6±8.7	3.5±8.9	EAR=2.0 ug
Vitamin C (mg)	83.3 ± 177.5	61.4 ± 77.4	54±151	27±59	EAR=75	63.2 ± 74.6*	69.3 ± 76.3**	42±84	32±49	EAR=60 mg
Vitamin D (ug)	5.5 ± 6.4	2.8 ± 4.1	na[4]	na[4]	AI=5	4.4 ± 5.8	3.1 ± 4.3	na[4]	na[4]	AI=5 ug
Vitamin E (mg)	17.8 ± 17.2	13.4 ± 14.9	na[4]	na[4]	EAR=12	17.0 ± 15.5	16.8 ± 19.7	na[4]	na[4]	EAR=12 mg

[1] DRI: dietary reference intakes; [2] AI: adequate intakes; [3] EAR: estimated average requirements; [4] na: not available; [5] nutrients added by mandatory fortification since 2004 (http://www.sajcn.co.za/index.php/SAJCN/article/viewFile/57/53); * p <0.05; ** p <0.01; *** p <0.0001 using the t-test between same age categories between the two studies.

Table 4.4.3 shows the NARs for the micronutrients for both studies. The mean NAR for vitamin A was above 100% in all the groups with the exception of older males in 1990. The mean NAR for vitamin B6 and calcium was less than 100% in the females for both studies. In the 1990 group, the females also had a NAR less than 100% for vitamin C, folate, riboflavin and thiamine. In males, the NAR for calcium was also below 100% in both study samples.

A linear regression model is presented in Table 4.4.4 with MAR as the dependent variable. Energy, protein and fat were all significant in the regression model. This was also the case for urbanisation which means that the more urbanised the participants were, the higher the MAR. So with more urbanisation, more macronutrients and micronutrients were being eaten and, thus, the higher the micronutrient adequacy of the diet. The AI also significantly correlated with MAR. The MAR will increase by 0.26% for every unit increase in BMI. Also, for every gram of protein the MAR will increase by 0.26%. Unexpectedly, for every gram increase in the intake of AS, the mean MAR will decrease by 0.04%, however, this was not significant. The average MAR was also found to be 2.86% higher for females than males.

Table 4.4.5 shows the number of food portions (based on diabetic exchanges) eaten per day from five food groups, viz. milk, meat, vegetables and fruit, cereal and fat.[147] The 2009 study had the highest number of portions consumed from the cereal group, where males had 9.45 and females had 8.73 mean portions per day, with a greater percentage coming from the refined cereal group. The second highest number of portions consumed was from the fat group, where males consumed 3.30 and females 4.2 mean portions per day. Vegetables and fruit were third on the list, where males consumed 2.78 and females 2.90 mean portions per day. This is followed by the meat and then lastly the dairy group where males and females were consuming less than one portion per day. In the 2009 study, milk products, the meat group, legumes, SF and brick margarine in males and females were found to be statistically lower than those in the 1990 study. This was also found in the cereal group, but just for the males. Despite this, the 2009 study for the males and females, showed significantly higher intakes of eggs, vitamin C rich fruits and vegetables and PUFA sources compared to the 1990 group. This was also found in the cereal group for females only. The percentage consumers for red meat, white meat, eggs, vegetables and fruit, and cereals increased from 1990 to 2009. For the milk and fat groups, the percentage consumers decreased.

Correlations of nutrients with duration of urbanisation (and the AI score) were done to test for significant associations) (Tables 4.4.6 and 4.4.7).

Table 4.4.3: Mean nutrient adequacy ratios of micronutrients of 25–64-year-old black adults in the CRIBSA study (2009) in Cape Town and comparison with 25–64-year-old black adults in the BRISK 1990 study[34]

	Males 2009		Males 1990		Females 2009		Females 1990	
Age groups: years	25-44	45-64	25-44	45-64	25-44	45-64	25-44	45-64
N	138	76	285	98	216	114	364	117
NAR Vitamin A (%)	182	187	116	63	213	156	112	115
NAR Vitamin B6 (%)	106	93	109	73	89	77	82	73
NAR Vitamin B12 (%)	488	478	345	190	289	198	180	175
NAR Vitamin C (%)	111	82	72	36	105	115	70	53
NAR Calcium (%)	38	32	53	52	31	35	34	36
NAR Folate (%)	105	319	68	57	90	297	48	46
NAR Iron (%)	165	139	123	123	187	181	117	117
NAR Niacin (%)	151	133	134	126	128	120	111	91
NAR Riboflavin (%)	158	101	109	100	165	116	89	89
NAR Thiamine (%)	120	100	110	100	113	114	100	89
NAR Zinc (%)	103	90	113	126	114	108	113	100
MAR[1]	77	71	na[2]	na[2]	75	76	na[2]	na[2]

NAR: Nutrient adequacy ratio: % of nutrient of the EAR value with 100% equalling 100% EAR value; MAR: mean adequacy ratio; MAR[1] for 1990 data calculated from [10]; na[2]: not available.

Table 4.4.4: Linear regression with mean adequacy ratio (MAR) of the diet as dependent variable for data from the 2009 study

Variable	Parameter estimate	P-value testing for the significance of the relationship in the regression model with MAR	Pearson correlation with MAR	P-value of correlation coefficient
Intercept	41.42	<0.0001***	-	
Urbanisation	0.03	0.0238*	0.1254	0.0034**
Asset index			0.1790	<0.0001***
BMI	0.23	0.0017**	0.1251	0.0035**
Age	-0.06	0.1394	-0.0885	0.0391*
Total protein	0.26	<0.0001***	0.5779	<0.0001***
Total energy			0.4421	<0.0001***
Total fat			0.3029	<0.0001***
Added sugar	-0.04	0.0038**	-0.210	0.6251
Gender	2.86	0.0110*	0.0207	0.6307
Adjusted R^2 = 0.4330				

* *p* <0.05; ** *p* <0.01; *** *p* <0.0001; BMI: body mass index.

Table 4.4.5: Mean food portion sizes (SD) consumed by the black participants in 2009 and 1990[34]

	Males 2009 n=215	Males 1990 n=441	Females 2009 n=329	Females 1990 n=542	% Consumers 2009	% Consumers 1990
Milk group	0.43 ± 0.77***	0.9±1.4	0.35 ± 0.64**	0.5±0.8	40.3	58
Milk	0.40 ± 0.76***	0.9±1.4	0.34 ± 0.63***	0.5±0.8	39.3	58
Cheese	0.03 ± 0.17	0.02±0.2	0.01 ± 0.12*	0.03±0.2	2.4	4
Meat group	2.12 ± 1.85**	2.6±2.3	1.68 ± 1.44*	1.9±1.6	82.7	88
Red meat	0.68 ± 1.02	0.8±1.7	0.48 ± 0.81	0.5±1.0	42.8	29
White meat	0.60 ± 0.92	0.7±1.0	0.54 ± 0.73	0.6±0.8	46.0	41
Eggs	0.73 ± 1.34***	0.4±1.0	0.53 ± 1.08***	0.3±0.8	24.4	16
Legumes	0.10 ± 0.49***	0.5±1.2	0.13 ± 0.42***	0.3±0.8	9.9	20
Vegetables & fruit	2.78 ± 2.58***	2.1±2.5	2.90 ± 2.52**	2.4±2.6	82.9	71
Vitamin C rich	0.57 ± 1.09***	0.2±0.6	0.68 ± 1.01***	0.2±0.6	47.8	21
Carotene rich	0.24 ± 0.65	0.2±0.5	0.34 ± 0.69**	0.2±0.6	27.9	18
Potato/sweet potato	1.08 ± 1.73	1.0±1.6	0.97 ± 1.42	1.0±1.5	48.7	45
Other veg/fruit	0.89 ± 1.72	0.7±1.5	0.94 ± 1.64	1.1±1.8	39.2	46
Cereal group	9.45 ± 4.78**	10.8±6.9	8.73 ± 3.31***	7.5±4.8	100.0	97
Unrefined	2.10 ± 2.40	2.0±3.7	1.59 ± 1.85**	1.2±2.1	55.0	37
Refined	7.34 ± 5.04**	8.7±7.0	7.14 ± 3.71**	6.4±4.9	97.2	92
Fat group	3.30 ± 5.14	3.4±3.9	4.20 ± 5.97	3.1±3.2	59.4	77
Saturated fat	0.10 ± 0.63***	0.8±2.1	0.32 ± 1.50***	0.7±1.7	5.5	26
Oil/tub margarine	1.89 ± 4.73***	0.5±1.4	2.50 ± 5.35***	0.6±1.3	28.1	29

[1] Portion sizes calculated according to diabetic exchange lists[146] * p <0.05; ** p <0.01; *** p <0.0001.

In Tables 4.4.6 and 4.4.7 data are provided on correlations of anthropometric and biochemical data in males and females. In males, BMI significantly correlated with energy, protein, animal protein, SF, SF %E, MUFA, and CHO, while in females, BMI significantly correlated with energy, dietary P:S ratio, CHO and AS. Energy and CHO are common to both sexes.

Diastolic BP significantly correlated negatively with fat and fat %E in males, whereas SBP is significantly negatively associated with fat, fat %E, PUFA, PUFA %E, and protein %E. In females, DBP negatively correlated with fat, fat %E, SF, MUFA, MUFA %E, and Na, while positively correlated with CHO %E. Systolic BP is negatively associated with fat, fat % E, MUFA, MUFA %E, PUFA, and Na, but is positively correlated with CHO %E. In males and females, fat, fat %E and E are common to both sexes in DBP and SBP, while in female both DBP and SBP are positively correlated with DBP and SBP.

With regard to blood TC in males, there are no significant correlations. However, in females, TC is significantly and negatively associated with E, fat, MUFA, CHO, and Na. In males, HDL-C is negatively significantly associated with CHO %E, AS, and calcium, while in females, it is positively correlated with protein %E and negatively with AS. There were no significant correlations in males for LDL-C. In females, LDL-C was negatively associated with energy intake. In males and females AS was negatively associated with HDL-C.

In males, WC was significantly and positively associated with energy, protein, animal protein, fat, SF, and CHO. In females, WC correlated negatively with the P:S ratio. In males, the HC was positively and significantly associated with energy, protein, animal protein, fat, SF, SF %E, MUFA, and CHO. In females, HC significantly correlated with energy, fat, MUFA, CHO, AS and Na. The WHR, significantly correlated negatively with PUFA and PUFA %E, in males. In females, the WHR significantly correlated negatively with the P:S ratio, CHO and AS. Both males and females had correlations in common for HC, i.e. energy, fat, MUFA and CHO.

With regard to the duration of urbanisation and AI in males, there were significant negative associations with plant protein and CHO and positive associations with animal protein, fat, fat %E, SF and SF %E, MUFA and MUFA %E. In females, there were positive correlations with protein, animal protein and SF and SF %E, potassium and calcium. There were negative correlations with P:S ratio, CHO and AS %E. Common positive correlations of urbanisation and AI, in males and females, were animal protein, SF, and SF %E. A common negative correlation with urbanisation and AI was CHO.

Table 4.4.8 presents a logistic regression model. Forward regression was done to identify the significant variables which were then entered into the model. In males, BMI was significantly associated with protein intake, while in females BMI was related to SF, SF %E, CHO, and CHO %E. In terms of the duration of urbanisation it can be noted that CHO %E, TS %E energy, calcium, animal protein and TC were significant in males. In females, protein %E, CHO %E, fat %E were significantly associated with greater urbanisation.

Table 4.4.6: Pearson correlations (r and p values) of the various anthropometric and biochemical variables with dietary parameters in males

	BMI	DBP	SBP	GLUCOSE	TC	HDL-C	LDL-C	TRIG	WC	HC	WHR	Urban	Asset Index
Energy (kJ)	0.16439*	-0.03782	-0.03635	0.20695**	0.04449	0.00509	0.03521	0.04129	0.17169*	0.18214**	0.07674	0.02192	0.01142
	0.0168	0.5849	0.5996	0.0026	0.5234	0.9419	0.6128	0.5538	0.0125	0.0080	0.2671	0.7515	0.8691
Protein(g)	0.22087**	0.05767	0.05332	0.09234	0.08653	0.00969	0.08764	0.03363	0.20647**	0.23783**	0.07396	0.10686	0.11186
	0.0012	0.4046	0.4410	0.1836	0.2140	0.8895	0.2070	0.6297	0.0026	0.0005	0.2849	0.1218	0.1052
Protein %E	0.11396	0.12902	0.14004*	-0.04175	0.06806	0.01885	0.06991	0.00529	0.08602	0.11658	0.01190	0.10317	0.09301
	0.0988	0.0614	0.0421	0.5484	0.3287	0.7869	0.3145	0.9395	0.2134	0.0912	0.8636	0.1352	0.1783
Plant protein (g)	0.02748	0.02640	0.03514	0.03119	-0.02150	-0.09994	0.00410	0.12025	0.07413	0.05209	0.05911	-0.13531*	-0.14890*
	0.6915	0.7030	0.6117	0.6539	0.7579	0.1509	0.9530	0.0836	0.2838	0.4516	0.3929	0.0497	0.0306
Animal protein (g)	0.20383**	0.06919	0.04523	0.09670	0.13340	0.07562	0.11651	-0.01457	0.17562*	0.21058**	0.05498	0.18829**	0.16903*
	0.0029	0.3171	0.5135	0.1636	0.0547	0.2776	0.0930	0.8346	0.0106	0.0021	0.4269	0.0061	0.0140
Fat (g)	0.12562	-0.10208	-0.15345*	0.20572**	0.07520	0.05189	0.05570	-0.00212	0.06607	0.15696*	-0.07719	0.15037*	0.17440*
	0.0686	0.1395	0.0258	0.0028	0.2804	0.4566	0.4231	0.9758	0.3396	0.0226	0.2643	0.0290	0.0112
Fat %E	0.07415	-0.13887*	-0.17948**	0.17285*	0.06240	0.02965	0.05703	-0.00109	-0.00105	0.09309	-0.11857	0.15630*	0.25486**
	0.2836	0.0439	0.0090	0.0123	0.3706	0.6707	0.4121	0.9875	0.9880	0.1780	0.0858	0.0232	0.0002
Saturated fat (g)	0.20721**	-0.05401	-0.09229	0.12507	0.07170	-0.04092	0.10684	0.02708	0.14179*	0.20019**	0.00024	0.13613*	0.27893***
	0.0025	0.4352	0.1817	0.0712	0.3034	0.5573	0.1236	0.6978	0.0396	0.0035	0.9972	0.0483	<.0001
Saturated fat %E	0.15697*	-0.07016	-0.08890	0.03263	0.05924	-0.09021	0.12708	0.03016	0.08995	0.13723*	-0.01605	0.13674*	0.33171***
	0.0226	0.3105	0.1984	0.6390	0.3953	0.1951	0.0667	0.6654	0.1931	0.0465	0.8167	0.0473	<.0001
MUFA (g)	0.15228*	-0.07046	-0.10505	0.18853**	0.07080	0.03158	0.05047	0.06055	0.11200	0.16738*	-0.01366	0.17512*	0.22499**
	0.0270	0.3084	0.1282	0.0063	0.3095	0.6507	0.4680	0.3850	0.1047	0.0149	0.8436	0.0108	0.0010
MUFA %E	0.10527	-0.08817	-0.10511	0.14819*	0.06680	0.00827	0.05821	0.08658	0.05738	0.10663	-0.03711	0.19374**	0.28346***
	0.1274	0.2021	0.1280	0.0322	0.3377	0.9056	0.4025	0.2137	0.4070	0.1226	0.5920	0.0047	<.0001
PUFA[7] (g)	-0.00346	-0.11191	-0.16365*	0.19292**	0.05214	0.10626	0.00560	-0.06553	-0.04771	0.05195	-0.14373*	0.07863	0.00165
	0.9601	0.1050	0.0174	0.0051	0.4545	0.1266	0.9358	0.3470	0.4906	0.4529	0.0370	0.2555	0.9809
PUFA %E	-0.05510	-0.13039	-0.17739**	0.18414**	0.02685	0.10850	-0.02247	-0.08544	-0.10529	-0.00943	-0.16975*	0.04331	0.02935
	0.4259	0.0586	0.0098	0.0076	0.7003	0.1188	0.7467	0.2198	0.1274	0.8917	0.0135	0.5315	0.6717
Diet P/S ratio	-0.12976	-0.05109	-0.08105	0.09985	0.00232	0.10884	-0.05078	-0.09726	-0.11803	-0.07847	-0.10295	-0.08163	-0.16151*
	0.0599	0.4604	0.2411	0.1503	0.9735	0.1176	0.4652	0.1622	0.0872	0.2564	0.1361	0.2377	0.0189

Table 4.4.6: Pearson correlations (r and p values) of the various anthropometric and biochemical variables with dietary parameters in males (continued)

	BMI	DBP	SBP	GLUCOSE	TC	HDL-C	LDL-C	TRIG	WC	HC	WHR	Urban	Asset Index
Cholesterol (mg)	0.11029	-0.01325	-0.04373	0.02451	0.03097	0.00109	0.05189	-0.04730	0.07729	0.10857	0.00725	0.00030	-0.05610
	0.1102	0.8483	0.5275	0.7246	0.6570	0.9875	0.4555	0.4975	0.2637	0.1159	0.9166	0.9965	0.4176
Carbohydrate (g)	0.14549*	-0.04792	-0.02042	0.15924*	-0.01233	-0.12353	0.03996	0.08072	0.16242*	0.13961*	0.10640	-0.15835*	-0.12547
	0.0347	0.4887	0.7681	0.0213	0.8597	0.0755	0.5657	0.2464	0.0182	0.0428	0.1234	0.0214	0.0689
Carbohydrate %E	-0.04927	0.01624	0.06349	0.08293	-0.08241	-0.16801*	-0.00609	0.05272	-0.03277	-0.09842	0.05899	-0.2115**	-0.25847**
	0.4766	0.8146	0.3588	0.2326	0.2367	0.0153	0.9303	0.4495	0.6360	0.1543	0.3939	0.0020	0.0001
Fibre (g)	0.00107	-0.00878	-0.01303	0.43462***	0.06317	-0.00852	0.05643	0.08972	-0.01762	0.04208	-0.08265	-0.04272	-0.00574
	0.9877	0.8992	0.8508	<.0001	0.3647	0.9028	0.4171	0.1975	0.7991	0.5433	0.2319	0.5372	0.9340
Added sugar (g)	0.10634	-0.11158	-0.04112	0.02853	0.01508	-0.15188*	0.11336	0.01447	0.08877	0.09540	0.04320	-0.11292	-0.03775
	0.1236	0.1060	0.5525	0.6818	0.8288	0.0285	0.1022	0.8357	0.1990	0.1674	0.5326	0.1019	0.5856
Sugar %E	0.04934	-0.07306	-0.00915	0.00496	0.00374	-0.15436*	0.10842	-0.00246	0.04835	0.01502	0.07330	-0.09313	-0.07098
	0.4759	0.2908	0.8949	0.9432	0.9573	0.0260	0.1181	0.9718	0.4848	0.8283	0.2892	0.1778	0.3048
Sodium	0.08776	-0.09001	-0.09333	0.09607	-0.02787	-0.06620	0.01147	-0.02891	0.07360	0.12474	-0.02128	0.04050	0.02433
	0.2042	0.1928	0.1768	0.1664	0.6895	0.3421	0.8691	0.6785	0.2873	0.0706	0.7586	0.5585	0.7253
Potassium	0.08455	-0.00171	-0.03899	0.31268***	0.01165	-0.01980	0.00546	0.07775	0.08377	0.10989	0.01438	0.07652	0.04352
	0.2213	0.9803	0.5732	<.0001	0.8674	0.7765	0.9375	0.2643	0.2256	0.1115	0.8355	0.2685	0.5295
Calcium	0.08643	-0.03290	-0.06360	0.03837	-0.07368	-0.17237*	0.01175	0.01233	0.06517	0.09430	-0.00225	-0.08303	0.06411
	0.2112	0.6347	0.3579	0.5812	0.2902	0.0128	0.8659	0.8598	0.3462	0.1724	0.9741	0.2298	0.3541

* Correlation significant, *p* <0.05; ** Correlation significant, *p* <0.01; *** Correlation significant, *p* <0.0001, BMI: Body mass index. DBP: Diastolic BP; SBP: Systolic BP; TC: Total cholesterol; HDL-C: High density lipoprotein cholesterol; LDL-C: Low density lipoprotein cholesterol; TRG: Triglycerides; WC: Waist circumference; HC: Hip circumference; WHR: Waist hip ratio; Urban: Duration of urbanisation

Table 4.4.7: Pearson correlations of the various anthropometric and biochemical variables with dietary parameters in females

	BMI	DBP	SBP	GLUCOSE	TC	HDL-C	LDL-C	TRIG	WC	HC	WHR	Urban	Asset Index
Energy (kJ)	0.11477*	-0.08264	-0.10157	0.17607**	-0.1449**	-0.09050	-0.11748*	-0.09328	0.09250	0.16140**	-0.08244	0.06355	0.06108
	0.0390	0.1383	0.0683	0.0015	0.0096	0.1072	0.0366	0.0968	0.0965	0.0036	0.1387	0.2540	0.2730
Protein (g)	0.05659	-0.03391	-0.04997	0.07568	-0.04042	0.02379	-0.02354	-0.06975	0.05786	0.08883	-0.03227	0.18954**	0.17292**
	0.3099	0.5437	0.3708	0.1742	0.4726	0.6725	0.6763	0.2148	0.2991	0.1105	0.5628	0.0006	0.0018
Protein % E	-0.00718	0.03279	0.03249	-0.03617	0.08018	0.11123*	0.07554	-0.00825	0.01579	-0.02118	0.06056	0.19620**	0.18326**
	0.8976	0.5570	0.5607	0.5165	0.1537	0.0475	0.1797	0.8834	0.7771	0.7040	0.2771	0.0004	0.0009
Plant protein (g)	-0.02568	-0.01361	-0.00285	0.08989	-0.05468	-0.00788	-0.03080	-0.08556	-0.02519	0.02932	-0.08757	0.00828	-0.07241
	0.6451	0.8074	0.9593	0.1063	0.3311	0.8887	0.5848	0.1279	0.6514	0.5990	0.1157	0.8820	0.1936
Animal protein (g)	0.08257	0.00144	-0.02399	0.06390	-0.04771	-0.01175	-0.02876	-0.03059	0.08253	0.07653	0.03216	0.16292**	0.15757**
	0.1381	0.9794	0.6676	0.2514	0.3964	0.8346	0.6099	0.5868	0.1383	0.1694	0.5641	0.0033	0.0045
Fat (g)	0.07862	-0.15932**	-0.17358**	0.04199	-0.11080*	-0.08086	-0.09045	-0.08740	0.09784	0.12293*	-0.01520	0.07805	0.09286
	0.1580	0.0041	0.0017	0.4513	0.0484	0.1502	0.1080	0.1199	0.0786	0.0269	0.7852	0.1610	0.0952
Fat % E	0.07162	-0.14418**	-0.15984**	-0.04905	-0.07837	-0.08932	-0.06006	-0.04617	0.10719	0.09266	0.04587	0.06450	0.08939
	0.1985	0.0095	0.0040	0.3788	0.1633	0.1119	0.2864	0.4120	0.0539	0.0959	0.4106	0.2470	0.1083
Saturated fat (g)	0.08693	-0.12971*	-0.08868	0.01349	-0.09044	-0.07163	-0.06833	-0.07645	0.09557	0.08436	0.03632	0.14714**	0.13120*
	0.1184	0.0197	0.1116	0.8089	0.1075	0.2027	0.2251	0.1738	0.0859	0.1297	0.5147	0.0080	0.0181
Saturated fat %E	0.09561	-0.10552	-0.04742	-0.05074	-0.03843	-0.05713	-0.01910	-0.03927	0.10674	0.05553	0.09965	0.15031**	0.15281**
	0.0857	0.0582	0.3957	0.3626	0.4947	0.3098	0.7347	0.4853	0.0549	0.3190	0.0733	0.0067	0.0058
MUFA (g)	0.08102	-0.14807**	-0.19481**	0.02970	-0.11357*	-0.06978	-0.10207	-0.08296	0.09850	0.12302*	-0.01638	0.10719	0.08351
	0.1456	0.0077	0.0004	0.5942	0.0430	0.2146	0.0695	0.1399	0.0766	0.0268	0.7690	0.0539	0.1336
MUFA %E	0.08504	-0.12463*	-0.18094**	-0.05341	-0.06961	-0.06264	-0.06221	-0.04804	0.12553*	0.10555	0.05429	0.11122*	0.09256
	0.1266	0.0251	0.0011	0.3379	0.2158	0.2654	0.2695	0.3932	0.0238	0.0577	0.3300	0.0455	0.0963
PUFA (g)	0.02892	-0.11009*	-0.13216*	0.04091	-0.07089	-0.05660	-0.05767	-0.05086	0.04664	0.08451	-0.04229	-0.03738	0.01432
	0.6040	0.0481	0.0175	0.4631	0.2074	0.3144	0.3060	0.3660	0.4028	0.1290	0.4481	0.5026	0.7973

Table 4.4.7: Pearson correlations of the various anthropometric and biochemical variables with dietary parameters in females (continued)

	BMI	DBP	SBP	GLUCOSE	TC	HDL-C	LDL-C	TRIG	WC	HC	WHR	Urban	Asset Index
PUFA %E	-0.01758	-0.07476	-0.10630	-0.01443	-0.06077	-0.06547	-0.05315	-0.00863	0.00380	0.03400	-0.04026	-0.09883	-0.05459
	0.7526	0.1801	0.0563	0.7958	0.2800	0.2444	0.3456	0.8781	0.9457	0.5420	0.4702	0.0757	0.3273
Diet P/S ratio	-0.11112*	-0.01170	-0.03709	-0.00261	-0.03872	-0.03226	-0.03715	-0.00909	-0.11476*	-0.04285	-0.12505*	-0.1862**	-0.21154**
	0.0457	0.8340	0.5066	0.9627	0.4914	0.5665	0.5099	0.8718	0.0390	0.4421	0.0244	0.0008	0.0001
Cholesterol (mg)	0.03782	-0.02461	-0.01957	0.12464*	-0.09135	-0.07424	-0.06357	-0.07211	0.03798	0.07900	-0.05051	0.04555	0.10235
	0.4975	0.6595	0.7261	0.0249	0.1039	0.1867	0.2591	0.1996	0.4957	0.1560	0.3648	0.4138	0.0658
Carbohydrate (g)	0.12534*	0.05123	0.03394	0.20342**	-0.11914*	-0.10160	-0.08859	-0.04584	0.05184	0.14870**	-0.13464*	-0.04760	-0.04126
	0.0240	0.3587	0.5434	0.0002	0.0337	0.0704	0.1154	0.4152	0.3523	0.0073	0.0153	0.3931	0.4592
Carbohydrate %E	-0.02819	0.13104*	0.14058*	0.04672	0.04142	0.01072	0.03387	0.06995	-0.09098	-0.05677	-0.07048	-0.1458**	-0.12778*
	0.6132	0.0185	0.0114	0.4020	0.4617	0.8490	0.5479	0.2135	0.1021	0.3083	0.2058	0.0085	0.0214
Fibre (g)	-0.05231	0.03380	0.04318	0.42026***	-0.01994	0.07323	-0.02848	-0.02889	-0.04042	-0.04854	-0.00720	0.02405	-0.03578
	0.3480	0.5450	0.4393	<.0001	0.7232	0.1928	0.6134	0.6078	0.4685	0.3839	0.8973	0.6663	0.5210
Added sugar (g)	0.14525**	0.05489	0.00604	-0.07085	-0.05956	-0.11302*	-0.02039	0.01966	0.04468	0.13374*	-0.13221*	-0.07304	-0.07327
	0.0088	0.3254	0.9138	0.2034	0.2897	0.0440	0.7176	0.7269	0.4228	0.0160	0.0173	0.1897	0.1884
Sugar %E	0.10116	0.07468	0.02687	-0.12111*	-0.01479	-0.09787	0.01603	0.01603	0.00021	0.06548	-0.10743	-0.11542*	-0.11102*
	0.0690	0.1806	0.6304	0.0293	0.7928	0.0814	0.7762	0.7762	0.9971	0.2398	0.0534	0.0378	0.0458
Sodium	0.06087	-0.17242**	-0.18676**	0.02902	-0.13965*	-0.06872	-0.10197	-0.10410	0.08097	0.13478*	-0.07223	0.14459**	0.12276*
	0.2746	0.0019	0.0007	0.6027	0.0127	0.2217	0.0698	0.0637	0.1459	0.0152	0.1947	0.0092	0.0271
Potassium	0.06091	0.03671	0.05246	0.37709***	-0.06944	0.02996	-0.06095	-0.06513	0.05930	0.04692	0.01824	0.12417*	0.06153
	0.2743	0.5109	0.3473	<.0001	0.2169	0.5945	0.2793	0.2468	0.2872	0.4000	0.7436	0.0254	0.2695
Calcium	0.07558	-0.04637	-0.01217	0.05092	-0.05888	-0.02020	-0.00747	-0.05685	0.04122	0.08370	-0.05161	0.13896*	0.15968**
	0.1747	0.4062	0.8275	0.3609	0.2952	0.7197	0.8946	0.3122	0.4596	0.1327	0.3544	0.0123	0.0040

* Correlation significant, p <0.05; ** Correlation significant, p <0.01; *** Correlation significant, p <0.0001; BMI: Body mass index. DBP: Diastolic BP; SBP: Systolic BP; TC: Total cholesterol; HDL-C: High density lipoprotein cholesterol; LDL-C: Low density lipoprotein cholesterol; TRG: Triglycerides; WC: Waist circumference; HC: Hip circumference; WHR: Waist hip ratio; Urban: Duration of urbanisation; E: Energy

Table 4.4.8: Logistic regression model resulting from forward regression to determine which variables to include in the model and then doing a regression with those variables

	Males				**Females**			
	Num var	Adjusted R^2	Sig F	Significant variables in model	Num var	Adjusted R^2	Sig F	Significant variables in model
Body mass index	10	0.0638	0.0094	Total Protein*	7	0.0642	0.0002	Saturated fat** Saturated fat %E** Carbohydrates** Carbohydrates %E*
Systolic blood pressure	7	0.0618	0.0054	Total fat % E** Total protein % E** Total cholesterol* Potassium* Kilojoules*	8	0.0507	0.0019	MUFA* PS Ratio *
Diastolic blood pressure	8	0.0378	0.0445	Total fat% E** Added sugar* Total protein% E*	8	0.0441	0.0044	Sodium** PUFA* Total carbohydrates*
Waist circumference	7	0.0599	0.0064	Total protein* PUFA*	12	0.0452	0.0089	MUFA %E* Total carbohydrates** MUFA* Total kilojoules*
Hip circumference	4	0.0582	0.0025	Total protein*	8	0.0685	0.0002	Total kilojoules* Total carbohydrates** Total carbohydrates %E*
Urbanisation	5	0.1053	<0.0001	Total carbohydrates% E** Total sugar %E* Calcium* Total animal protein** Total cholesterol**	15	0.0766	0.0005	Total protein %E** Total carbohydrates %E* Total fat %E*
Asset index	13	0.1964	<0.0001	Total carbohydrates %E** Total cholesterol** Total protein* Total fibre**	8	0.0893	<0.0001	PS ratio* PUFA ** Total protein %E* Sodium* Plant protein*

* *p* <0.05; ** *p* <0.01; *** *p* <0.0001.

4.5 HYPOTHESIS

With regard to the null hypothesis

"There are no significant differences in dietary intake (energy, macro-, micro-nutrients, and food portions) data of black adults in 2009 compared to 1990." This hypothesis is rejected since the data presented in the results section (Tables 4.4.1–4.4.5) clearly showed that there were significant differences in energy, macro- and micronutrient intakes and food potions between the two studies.

"There are no significant differences in association between energy and nutrients with urbanisation and assets of black adults in 2009." This hypothesis is rejected since the tables presented in the results section (Table 4.4.4) showed that there were significant associations.

"There are no significant associations among dietary intake data with anthropometry, (BMI, WC, HC, WHR), BP, biochemical data (TC, HDL-C, LDL-C, TG and glucose); and BP of black adults in 2009." Tables 4.4.6 and 4.4.7 show that there were many significant correlations between dietary intake and anthropometry, BP, and biochemical data.

CHAPTER 5: DISCUSSION

The first phase of the study indicated that the CRIBSA dietary energy intake was low in comparison with four other studies in urban black South Africans which have been published since 2000. In the THUSA[40] study in North West, middle-class urban males had a mean intake of 9897 kJ and females 8010 kJ compared to 5760 kJ in females and 6516 kJ in males in the CRIBSA study. The mean energy intake was 12 301 in females and 15 485 in males in the PURE study (unpublished data), in urban areas in the North West province. In a study among urban black females in the Vaal region, energy intakes of 10 093 for females were shown. With regard to mean energy intakes in urban areas of Free State, urban males had an intake of 8630 kJ and females had a mean intake of 7755 kJ.[38]

The highest mean energy intakes were seen in the PURE study, therefore, the fact that frequency questionnaires do tend to over-report, while the 24HR tends to under-report was taken into consideration.[146] However, since the other two studies mentioned above also had higher mean energy intakes, despite using the 24HR, it was decided to remove the under-reporters. This was further supported by the high prevalence of obesity found in the CRIBSA participants, with 12.3% males and 62.6% females having a BMI ≥30 kg/m^2. Once the under-reporters were removed the findings were more in line with those of the North West, Vaal and Free State studies.[41,148] Mean energy intakes changed from 7666–8557 kJ in males and from 7104–7619 kJ in females after the removal of under-reporters in the present study.

With regard to the comparison of the CRIBSA data with the BRISK data there were some significant changes indicative of the nutrition transition having taken place over the past 20 years. In CRIBSA males, there were significant increases in fat and fat %E (25–44 years), PUFA, PUFA %E, P:S ratio, and TC, while the amount of CHO, protein %E, and animal and plant protein decreased significantly. In females, energy intake, animal protein (25–44 years), fat, fat %E, SF, SF %E (25–44 years) MUFA, PUFA, P:S ratio, TC, and AS increased significantly compared to the BRISK participants. The females appeared to have had a more atherogenic diet than the males since their AS intake had also increased and their CHO intake had not decreased. These results, overall, mimic those of a typical Western diet which is high in energy intake, animal protein, fat, SF, AS, and low in CHO intake.[73]

If one evaluates the actual percentages of macronutrients to energy intake, it becomes noticeable that since 1990 the nutrition transition has taken place, particularly in females. Fat intakes as % energy were higher in CRIBSA males at 27.2–32% compared to 23.8–25.9% in BRISK males. In CRIBSA females, this was similarly higher at 32.6–33.4% compared to 26–27% in BRISK females.

Another large difference occurred in the CHO intake, which decreased significantly in CRIBSA males compared to BRISK males (53.2–57.4% vs. 59.2–61.3%). Similar results were found in the females, once again emphasizing the changes which took place in fat and CHO intakes. These results are also supported by our results (before removal of under-reporters), since the same trends were observed. Significant differences were noted with increased years of life spent in the city with increased mean values of fat, SF, MUFA, animal protein and decreased mean values of carbohydrate. Similarly, sugar intakes decreased with increased years of life spent in the city.

The CRIBSA fat %E values were higher than those found in the PURE and THUSA studies in both males and females, and CHO values were lower emphasizing perhaps an even further transition with regard to the CRIBSA study being a more recent study.[40]

Notably, the energy intake in males was similar in 1990 and 2009, which may account for the obesity prevalence of 9.5% being similar in the two studies (obesity prevalence in the larger sample, as well as the sample after under-reporters were removed).[118] In contrast, energy intake in females increased from 1990 to 2009 as did obesity levels.[34] However, energy intake was within the normal range and does not reflect the extremely high prevalence of obesity among females in this population (1990: 41.7%; 2009: 61.5%).

When examining the micronutrient intakes of the CRIBSA participants, it is clear that there are significant improvements in many micronutrient intakes compared to the BRISK participants which include iron, vitamin A, thiamine, riboflavin, vitamin B6, niacin, and folate. This is a clear indication that the fortification of maize and wheat flour with these micronutrients has improved their intakes. Mandatory fortification of food became official in 2003.[149] However, it should be noted that some micronutrients are still below the DRI, and notably calcium, zinc, vitamin C and vitamin D. This is also illustrated by the NARs for calcium, zinc, vitamin C and vitamin B6, which lie below 100%. Overall the MAR was between 71–77% where ideally it should have been 100%. The same pattern of dietary deficiencies has also been noted in the studies by Tydeman[41] and Oldewage-Theron and Kruger[148] among their black adult participants. Similar micronutrient deficiencies were also noted in the Phase 1 data, which again point to the reliability of the findings.

The effects of the nutrition transition are less noticeable when portion sizes of food are examined and compared to 1990 data. There were significant decreases in milk, meat, and legume intakes in males and females compared to the BRISK participants. However, fruit and vegetable consumption increased (although not much) and specifically vitamin C rich ones. While cereals decreased in males, they increased significantly in females, with regard to refined and unrefined products. There was also a significant increase in PUFA products such as oil and margarines in males and females. As found by Bourne et al. (1993)[34] in the BRISK participants, the dietary pattern had a very narrow range of foods, with insufficient intake of dairy products and fruits and vegetables. The 1990 study found that with increased time spent in the city, dairy consumption decreased by 33% and cereal intake by 26%, while fruit and vegetable intake increased by 19%, fats by 8%, and meat by 14%. In the CRIBSA study, fruit and vegetable intake was higher than in the 1990 study. However, dairy intake was even lower than in 2009. Cereal intake decreased in males in 2009 since 1990.

The low consumption of dairy products is of great concern, since dairy items are the main source of calcium and riboflavin in many diets. Unfortunately, calcium is not added to the fortification mix of maize and wheat flour, while riboflavin is. Since calcium is associated with bone health, particularly in the elderly, it is important to consider the promotion of dairy products to adults as well as children. The low intake of fruits and vegetables in the CRIBSA study is also of concern since fruits and vegetables have been found to be protective towards the development of NCD. They have been found to lower the risk of stroke, IHD, colorectal cancer and stomach cancer.[150,151,152] Furthermore, they contribute to cardio-metabolic health by virtue of their fibre, potassium and phyto-nutrients.[151,152]

With regard to the duration of urbanisation and AI in males, there were significant negative associations with plant protein and CHO, and positive associations with animal protein, fat, fat %E, SF and SF %E, MUFA and MUFA %E. In females, there were positive correlations with protein, animal protein and SF and SF %E, potassium and calcium. There were negative correlations with P:S ratio, CHO and AS %E. With the exception of sugar in females, the correlations were all in line with the effects on the nutrition transition where there were negative associations with CHO and plant proteins and positive associations with animal protein, SF, and fat %E.

The results of the dietary intake in the CRIBSA study are in keeping with the nutritional transition occurring in urban centres in SA. This has also been shown in the THUSA and PURE studies, with a greater westernisation of the diet as people move from rural to urban areas and from low to high SES. This is unlike rural areas where commonly traditional diets are low in fats (<25% of energy intake) and sugar (<10% energy), and high in CHO (>65% energy). In urban centres there is a shift with the adoption of a more western type of diet, which is higher in fat consumption (>30% of energy intake) and lower in CHO (<65%), as mirrored in this study. The typical Western diet is associated with more degenerative diseases, while the traditional diet is not. These findings are further illustrated by the significant positive correlations between greater duration of urbanisation with total fat, SF, MUFA and fat as a percent of energy intake. These correlations were also significant when done with the AI. Socio-economic advancement is known to also be associated with an increased intake of energy-dense foods associated with a Western lifestyle. A further reinforcement of this transition is the differences observed in fat intake between 1990[34] and 2009. This rise is caused by an increase in PUFA, since SF intake remained similar in the two studies, although rising significantly in the younger females.

Data from the correlations between the dietary intake data and anthropometry and biochemical indices can be explained. Males and females had a significant correlation among BMI and energy and CHO. This serves as a test of the reliability of the data, since it is expected that BMI will increase as energy and CHO increase. An interesting and expected significant negative correlation was between AS and HDL-C in both males and females. This is in line with the expectation that AS is associated with an atherogenic diet and as such will result in a decrease in HDL-C. More expected correlations emphasizing the detrimental aspects of the westernisation of the diet were the significant positive correlations between urbanisation and AI with protein, SF and SF %E, and negative associations with CHO. These stem from the fact that people with affluence and time lived in the city consume more meat, which increases their nutrient intakes.

The data from the logistic regression model support those of the correlations. However, two interesting aspects should be noted with regard to the duration of urbanisation, males and females have different significant variables. For example, in males the significant variables are CHO %E, TS %E, calcium, animal protein and cholesterol, while in females they are protein %E, CHO %E and fat %E. Similarly, this is found with the AI, with males having significant outcomes with CHO %E, cholesterol, protein and fibre, while in females these are P:S ratio, PUFA, protein %E, Na and plant protein. These differences between males and females were also observed in the correlations with the BP and biochemistry results, and imply that males and females are having some significant differences in dietary intake. Therefore, it would be worthwhile to further explore this issue.

5.1 LIMITATIONS OF THE STUDY

In this study a single 24HR was used in an attempt to recreate the same methodology used in the BRISK study in 1990. However, this method is known to underestimate dietary intake. A further limitation which needs to be kept in mind is the fact that because only one 24-hour recall was used it was not possible to determine within-person variation and hence usual intakes. Thus nutrient inadequacies which do not depend on the mean intake but rather on the variation of the usual intake distribution could not be adequately portrayed. Another limitation was the fact that the researcher had to use mean dietary intakes despite the data being non-parametric since mean intakes were presented in the 1990 data.

Furthermore, the researcher did not have access to the 1990 data which meant that she was unable to do certain statistical calculations. Another limitation was that the original sample which included the under-reporters decreased substantially after applying the Goldberg method.[148] Hence, one is unable to generalise the results. The mean energy intake levels of the females are difficult to explain because of their high prevalence of obesity. However, it is also possible that low PA levels may explain these, since a low prevalence of PA in black females was also found in the SANHANES.[8]

CHAPTER 6: CONCLUSIONS

The nutrient intakes demonstrate that while certain changes have taken place between 1990 and 2009, the dietary pattern regarding foods eaten remains poor. The diet has become more urbanised and atherogenic with regard to distribution of fat and CHO intake, while the consumption of certain food groups has remained low, such as the poor consumption of dairy products and low intake of fruit and vegetables. On the other hand, low micronutrient intakes have decreased over the two time periods as a result of mandatory fortification of staple foods in 2003.

Interventions are urgently needed to combat the shift towards the diet continuing to become more atherogenic and to improve the consumption of priority food groups, such as dairy products and fruit and vegetables. The introduction of “salt legislation” and the “sugar tax on sugar sweetened beverages” is expected to also contribute to improved health in the population. However, it is also clear that males and females may require different approaches to changing their dietary intake and/or lifestyles, particularly in terms of the impact of the nutrition transition. Thus, interventions to reduce the prevalence of obesity and NCD are vital in addressing this rising prevalence.

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ADDENDUM 1: INFORMATION FORM

The Medical Research Council

Chronic Diseases of Lifestyle Unit

P O Box 19070, Tygerberg 7505, South Africa
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No 3

Information regarding the CRIBSA study:

Cardiovascular Risk in Black South Africans

Purpose:

Diabetes, high blood pressure or high high and cholesterol problems or high fat levels in the blood seem to be more common in the community than they were 10 or 20 years ago. This is most likely a result of both environmental and genetic factors. Environmental factors are those factors related to your lifestyle such as your diet and your physical activity levels. Genetic factors are those factors you "inherit" from your parents and grandparents. These conditions can lead to further health problems such as problems with eyesight, the heart and strokes, but the chance of having these problems can be lessened by treatment.

The aim of the study is to investigate how many people in the community have high blood sugar, high blood pressure, high cholesterol and other risk factors for heart disease and stroke. This information will help with the planning of health services

Who can participate?

People between the ages of 25-75 will be randomly selected to participate in this study.

Benefits:

You will receive your own results informing you whether you have diabetes, high blood pressure, high cholesterol or high blood fats as well as your body size measurements. You will be referred to your doctor or clinic if this is necessary.

The findings of this research can be used to help prevent, treat and manage diseases associated with heart disease and stroke in South African people.

Procedures and potential risks:

Procedures:

1) Questionnaire:
We will ask you questions about your background like your education and housing, your family history, personal health, dietary intake and physical activity and health habits.

2) *Body Measurements:*

Basic body measurements will be taken such as weight, height, waist and hip circumference.

3) *Blood Pressure Measurement:*

After a 5 minute relaxation period, blood pressure will be measured 3 times in a row, separated by 5 minutes between readings using a standard blood pressure monitor.

4) *Blood Sampling:*

After an overnight 10 hour fast (where you do not eat or drink anything except water after your evening meal, the night before the blood sampling, and miss your breakfast the day of the test), we will place a sterile little tube in a vein in your arm and take 15 mls (3 teaspoons) of blood from this. We will give you a cup of sugar water to drink and then we will take two more blood samples (each 2 teaspoons) over the next two hours. Taking the blood sample may cause a little discomfort at the site but there are no risks for this test, other than those associated with routine blood sampling. All procedures will be supervised and carried out by appropriately trained medical personnel who will use techniques to minimise any risks of infection. This test is used routinely for medical purposes. The blood sample will be used to determine your blood sugar, insulin, cholesterol and other additional factors that may help us learn more about diabetes.

Assurances:

Participation in this research study is absolutely voluntary. You do not have to take part if you do not want to. You may withdraw at any time without stating a reason and without prejudice. The doctor or researcher can also withdraw you from the study. You will be provided with all your own results.

Your own results will only be given to you. All records will be kept in a locked room and in a secure computer database in the research unit. Your name will not be used in any publication of the results.

The University of Cape Town and its team of researchers, who are working under the mandate of the University, will be responsible for treating any adverse or untoward events arising from participation in this research study.

If any medical problems are identified during this study you will be referred to the public health system for further help.

Any required transport to the testing station will be arranged by the researchers and be at no cost to you.

Thank you for your participation. Please contact me if you would like to ask any questions or you experience any problems during or after the tests.

Professor N. S. Levitt
Department of Medicine
University of Cape Town

Phone: 021 404 5007

Dr Nasheeta Peer
Chronic Diseases of Lifestyle Unit
Medical Research Council

Phone: 021 938 0811

ADDENDUM 2: INFORMED CONSENT

CONSENT TO PARTICIPATE IN RESEARCH STUDY

Cardiovascular Risk in Black South Africans (CRiBSA) Study

Investigators: Dr N Peer, Prof NS Levitt, Prof K Steyn, Dr N Steyn, Prof VE Lambert,

Dr C Lombard

STUDY NUMBER				

Dear Participant

You are being asked to participate in a research study.

You have heard and understood an explanation of the research project and have read a written explanation of what is required. Confidentiality will be adhered to at all times.

You are free to refuse to participate or can **withdraw** permission to take part at any time and you need not answer all the questions during the interview. Any questions that you may have at any time will be answered.

You may contact **Dr Nasheeta Peer** at telephone (021) **938 0811** any time you have questions about the research.

Your participation in this research is voluntary, and you will not be penalised or lose benefits if you refuse to participate or decide to stop at any stage.

If you agree to participate, you will be given a signed copy of this document and a written summary of the research.

1.) I hereby consent to participate in this research study.

.... ………………..

Signature of participant Date

To my knowledge, consent was given willingly and with full understanding.

.... ………………..

Signature of interviewer Date

.... ………………..

Signature of Witness Date

DNA will be isolated and stored for future analyses of genes related to cardiovascular disease (CVD) and its risk factors. This is a new area of study and not all genetic aspects of CVD are currently known. These tests are done anonymously and will not be linked back to any specific person.

2.) *I also give permission that my DNA may be isolated and stored for future analysis, as explained to me.*

.... ………………..

Signature of participant Date

To my knowledge, consent was given willingly and with full understanding.

.... ………………..

Signature of interviewer Date

.... ………………..

Signature of Witness Date

ADDENDUM 3: ETHICS APPROVAL

MAILED

Department of Human Nutrition

3rd Floor

Clinical Building

Dear Mrs Jaffer

Mrs N Jaffer

ETHICS REFERENCE NO: N11/06/189

25 July 2011

A panel of the Health Research Ethics Committee reviewed this project on 5 July 2011; the above project was approved on

condition that further information is submitted.

This information was supplied and the project was finally approved on 25 July 2011 for a period of one year from this date.

This project is therefore now registered and you can proceed with the work.

Please quote the above-mentioned project number in ALL future correspondence.

Please note that a progress report (obtainable on the website of our Division: www.sun.ac.za/rds should be submitted to the

Committee before the year has expired. The Committee will then consider the continuation of the project for a further year (if

necessary). Annually a number of projects may be selected randomly and subjected to an external audit.

Translations of the consent document in the languages applicable to the study participants should be submitted.

Federal Wide Assurance Number: 00001372

Institutional Review Board (IRB) Number: IRB0005239

The Health Research Ethics Committee complies with the SA National Health Act No.61 2003 as it pertains to health research

and the United States Code of Federal Regulations Title 45 Part 46. This committee abides by the ethical norms and

principles for research, established by the Declaration of Helsinki, the South African Medical Research Council Guidelines as

well as the Guidelines for Ethical Research: Principles Structures and Processes 2004 (Department of Health).

Please note that for research at a primary or secondary healthcare facility permission must still be obtained from the relevant

authorities (Western Cape Department of Health and/or City Health) to conduct the research as stated in the protocol. Contact

persons are Ms Claudette Abrahams at Western Cape Department of Health (healthres@pgwc.gov.za Tel: +27 21 483 9907) and Dr Hélène Visser at City Health (Helene.Visser@capetown.gov.za Tel: +27 21 400 3981). Research that will be conducted at any tertiary academic institution requires approval from the relevant hospital manager. Ethics approval is required BEFORE approval can be obtained from these health authorities.

Approval Date: 25 July 2011 Expiry Date: 25 July 2012

RE : APPROVAL

Comparison of Dietary Intake and Nutritional Status of the Urban Black Population in the Cape Peninsula in 1990 with

2008/09

25 July 2011 12:00 Page 1 of 2

Fa k ul t e i t Ge s on dh e id swe t e n s k ap pe · Fa c ul t y o f He a l th Sc i enc e s

Verbind tot Optimale Gesondheid · Committed to Optimal Health

Afdeling Navorsingsontwikkeling en -steun · Division of Research Development and Support

Posbus/PO Box 19063 · Tygerberg 7505 · Suid-Afrika/South Africa

Tel.: +27 21 938 9075 · Faks/Fax: +27 21 931 3352

MS CARLI SAGER

Yours faithfully

RESEARCH DEVELOPMENT AND SUPPORT

Tel: +27 21 938 9140 / E-mail: carlis@sun.ac.za

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25 July 2011 12:00 Page 2 of 2

Fa k ul t e i t Ge s on dh e id swe t e n s k ap pe · Fa c ul t y o f He a l th Sc i enc e s

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ADDENDUM 4

RISK IN BLACK SOUTH AFRICANS (CRiBSA)

QUESTIONNAIRE

SECTION 1: GENERAL INFORMATION	*Office use*
STUDY NUMBER:	4
INTERVIEWER'S NAME	
AREA/TOWNSHIP	
NAME OF PARTICIPANT..	
ADDRESS OF PARTICIPANT...	
....	
TELEPHONE	
2ND CONTACT PERSON'S DETAILS:	
NAME..	
ADDRESS...	
....	
TELEPHONE	
DATE OF INTERVIEW: D D M M Y Y Y Y	16
GENDER: Male 1 / Female 2	
AGE AT LAST BIRTHDAY	
DATE OF BIRTH: D D M M Y Y Y Y	27

SECTION 2: MIGRATORY HISTORY (to calculate % life spent in urban setting)	Office use
2E. Have you spent any time away from the city, for at least a full year without a break, since you first arrived or since birth? Yes 1 / No 2*	37
2F. If YES:	

Place:	Length of time in Years
1.	
2.	
3.	
4.	
5.	
6.	
Total:	

Office use: 39

***2E. If NO, go to Section 3**

SECTION 3: SOCIO-DEMOGRAPHIC INFORMATION	*Office use*

3A. What is the highest level of education that you have achieved? 40

Never went to school 1	Grade 1-7 (Primary school) 2	Grade 8-12 (High school) 3	Tertiary/Diploma 4

3B. Are you …

Employed 1	Unemployed 2	A full-time homemaker 3	A pensioner 4	On a disability grant 5	A student 6

3C. What type of housing do you live in?

Built formal unit/ privately owned 1	Council / core house 2	Informal shack / shelter/hostel/other 3

3D. How many rooms does your house have (excluding bathroom & kitchen)? ……………………

3E. How many people are living with you in your house? …………………… 46

3F. Does your household have:

1)	Electricity?	Yes 1	No 2
2)	A radio?	Yes 1	No 2
3)	A television?	Yes 1	No 2
4)	A telephone (landline or cellular)?	Yes 1	No 2
5)	A refrigerator?	Yes 1	No 2
6)	A personal computer (PC)?	Yes 1	No 2
7)	A washing machine?	Yes 1	No 2
8)	Tap inside the house?	Yes 1	No 2
9)	A toilet inside?	Yes 1	No 2
10)	A motor car?	Yes 1	No 2
11)	A bicycle (adults)?	Yes 1	No 2

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SECTION 4: SELF-REPORTED MEDICAL HISTORY	*Office use*
STUDY NUMBER:	4

4A. Would you say your health is poor, average, good, or very good/excellent?

Poor 1	Average 2	Good 3	Very good/excellent 4

4B. Do you personally think that you are underweight, normal weight or overweight?

Underweight 1	Normal weight 2	Overweight 3	Don't know 9

4C. Has a **doctor** or **nurse** or **health worker** at a **clinic** or **hospital told you** that you have or hav had

any of the following conditions?

1)	High blood pressure	Yes 1	No 2	Don't know 9
2)	Heart attack or angina (chest pains)	Yes 1	No 2	Don't know 9
3)	Stroke	Yes 1	No 2	Don't know 9
4)	High blood cholesterol or fats in the blood	Yes 1	No 2	Don't know 9
5)	Diabetes or blood sugar	Yes 1	No 2	Don't know 9
6)	Diabetes during pregnancy	Yes 1	No 2	Don't know 9

12

4D. During the past 12 months have you been tested/checked for:

1)	Hypertension/high blood pressure	Yes 1	No 2
2)	Diabetes	Yes 1	No 2

4E. During the past 12 months have you seen a traditional healer for:

1)	Hypertension/high blood pressure	Yes 1	No 2
2)	Diabetes	Yes 1	No 2

4F. Are you currently taking any herbal or traditional remedy for:

1)	Hypertension/high blood pressure	Yes 1	No 2
2)	Diabetes	Yes 1	No 2

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SECTION 5: MEDICATION		*Office use*	
5A. Who pays for **most** of the medication, prescribed by a doctor or nurse, that you use?			
READ THE ANSWER CATEGORIES TO THE RESPONDENT			
Respondent	1		
Family	2		
Medical aid	3		
Provided at clinic or public hospital	4		
Employer	5		
Other (specify) …………………………………………………………………………..	6		19
5B. Do you use any medicine regularly or daily that a doctor or nurse has prescribed?			
	Yes 1 / No 2		
5C. How many different medicines do you use regularly or daily?	No. of medicines:		22

SECTION 5: MEDICATION	*Office use*
STUDY NUMBER:	☐☐☐☐ 4
5D. RECORD ALL THE DRUGS BROUGHT IN BY THE RESPONDENT:	
1)	☐☐☐☐☐☐☐ 11
2)	☐☐☐☐☐☐☐ 18
3)	☐☐☐☐☐☐☐ 25
4)	☐☐☐☐☐☐☐ 32
5)	☐☐☐☐☐☐☐ 39
6)	☐☐☐☐☐☐☐ 46
7)	☐☐☐☐☐☐☐ 53
8)	☐☐☐☐☐☐☐ 60
9)	☐☐☐☐☐☐☐ 67
10)	☐☐☐☐☐☐☐ 74
11)	☐☐☐☐☐☐☐ 81

SECTION 6: FAMILY MEDICAL HISTORY				*Office use*
STUDY NUMBER:				4
6A. Do you have a **close blood relative** (father, mother, brother, sister / child) who has ever bee diagnosed: by a doctor or nurse with any of the following conditions?				
1) High blood pressure	Yes 1	No 2	Don't know 9	
2) Heart attack/angina/chest pain when exertir himself/herself	Yes 1	No 2	Don't know 9	
3) Stroke	Yes 1	No 2	Don't know 9	
4) Diabetes	Yes 1	No 2	Don't know 9	
6B. Nowadays in South Africa far more young people die, or become very ill than before.				
Has this happened in your family?	Yes 1	No 2		
6C. Do you have to take care of a child whose parents are ill or have died?	Yes 1	No 2		
6D. If YES, has this made it more difficult for you to care for your own health?	Yes 1	No 2		
6E. Does anyone in your household have HIV/AIDS?	Yes 1	No 2	Don't know 9	12

SECTION 7: PSYCHOSOCIAL

Office use

STUDY NUMBER: 4

Now I am going to ask you questions relating to certain aspects of your life. Some questions may seem the same but please bear with me because they are not exactly the same.

A) ORIENTATION TO LIFE QUESTIONNAIRE (SOC-13)

Below is a series of questions relating to various aspects of our lives. For each question, you will first select the response which most closely resembles how you feel. Then, on a scale from 1 to 7, you will select the number closest to that response which best describes how you feel. You can choose any number between 1 and 7. Please give only one answer to each question.

1) Do you have the feeling that you don't really care about what goes on around you?

Very seldom or never				Very often		
1	2	3	4	5	6	7

5

2) Has it happened in the past that you were surprised by the behaviour of people whom you thought you knew well?

Never happened				Always happened		
1	2	3	4	5	6	7

3) Has it happened that people whom you relied/depended on disappointed you?

Never happened				Always happened		
1	2	3	4	5	6	7

4) Until now your life has had:

No clear direction or purpose at all				Very clear direction and purpose		
1	2	3	4	5	6	7

8

5) Do you have the feeling that you're being treated unfairly?

Very often				Very seldom or never		

1	2	3	4	5	6	7

6) Do you have the feeling that you are in an unfamiliar situation and don't know what to do?

Very often			Very seldom or never			
1	2	3	4	5	6	7

7) Doing the things you do every day is:

A source of deep pleasure and satisfaction			A source of frustration and boredom			
1	2	3	4	5	6	7

8) Do you feel confused or have very mixed-up feelings and ideas?

Very often			Very seldom or never			
1	2	3	4	5	6	7

12

9) Does it happen that you have feelings inside that you don't like or would rather not feel?

Very often			Very seldom or never			
1	2	3	4	5	6	7

10) Many people—even those who are confident and successful—sometimes feel like losers in certain situations. How often have you felt this way in the past?

Never			Very often			
1	2	3	4	5	6	7

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SECTION 7: PSYCHOSOCIAL	Office use

11) When something happened, have you generally found that:

You over-estimated or underestimated its importance?				You saw things in the right perspective?			
1	2	3	4	5	6	7	15

12) How often do you have the feeling that there's little meaning in the things you do in your daily life?

Very often				Very seldom or never		
1	2	3	4	5	6	7

13) Sometimes people have strong feelings that they cannot keep under control. How often do you have feelings that you're not sure you can keep under control?

Very often				Very seldom or never		
1	2	3	4	5	6	7

B) LOCUS OF CONTROL

To which extent do you agree or disagree with the following statements about your own life:

	Strongly disagree	Disagree	Neutral	Agree	Strongly agree	
1) In your general work, you feel you have control over what happens in most situations	1	2	3	4	5	18
2) You feel what happens in your life is often determined by factors beyond your control	1	2	3	4	5	
3) Over the next 5-10 years, you expect to have more positive than negative experiences	1	2	3	4	5	
4) You often have the feeling you are being treated unfairly	1	2	3	4	5	
5) In the past 10 years your life has been full of changes without you knowing what will happen next	1	2	3	4	5	
6) You gave up trying to better your life a long time ago	1	2	3	4	5	23

C) LIFE EVENTS QUESTIONNAIRE

Have any of the following life events or problems happened to you during the last 6 months? What about more than 6 months ago? If so, please also rate the impact on you.

1) You yourself suffered a serious illness, injury or an assault		Yes 1	No 2 **go to next question**	
1a) Did this occur in the past 6 months?			Yes 1 | No 2	
If **yes**, Impact:	None 3	Some 4	Significant 5	
1b) Did this occur more than 6 months ago?			Yes 1 | No 2	
If **yes**, Impact:	None 3	Some 4	Significant 5	
2) A serious illness, injury or assault happened to a close relative of yours		Yes 1	No 2 **go to next question**	29
2a) Did this occur in the past 6 months?			Yes 1 | No 2	
If **yes**, Impact:	None 3	Some 4	Significant 5	
2b) Did this occur more than 6 months ago?			Yes 1 | No 2	
If **yes**, Impact:	None 3	Some 4	Significant 5	33

SECTION 7: PSYCHOSOCIAL					*Office use*	
3) Your parent, child or spouse died		Yes 1	No 2 **go to next question**			34
3a) Did this occur in the past 6 months?			Yes 1	No 2		
If **yes**, Impact:	None 3	Some 4	Significant 5			
3b) Did this occur more than 6 months ago?			Yes 1	No 2		
If **yes**, Impact:	None 3	Some 4	Significant 5			
4) A close family friend or another relative (aunt, cousin, Grandparent) died		Yes 1	No 2 **go to next question**			39
4a) Did this occur in the past 6 months?			Yes 1	No 2		
If **yes**, Impact:	None 3	Some 4	Significant 5			
4b) Did this occur more than 6 months ago?			Yes 1	No 2		
If **yes**, Impact:	None 3	Some 4	Significant 5			
5) You had a separation caused by marital difficulties		Yes 1	No 2 **go to next question**			44
5a) Did this occur in the past 6 months?			Yes 1	No 2		
If **yes**, Impact:	None 3	Some 4	Significant 5			
5b) Did this occur more than 6 months ago?			Yes 1	No 2		
If **yes**, Impact:	None 3	Some 4	Significant 5			
6) You broke off a steady relationship		Yes 1	No 2 **go to next question**			49
6a) Did this occur in the past 6 months?			Yes 1	No 2		
If **yes**, Impact:	None 3	Some 4	Significant 5			
6b) Did this occur more than 6 months ago?			Yes 1	No 2		
If **yes**, Impact:	None 3	Some 4	Significant 5			

7) You had a serious problem with a close friend, neighbour or relative of yours		Yes 1	No 2 **go to next question**		54
7a) Did this occur in the past 6 months?			Yes 1	No 2	
If **yes**, Impact:	None 3	Some 4	Significant 5		
7b) Did this occur more than 6 months ago?			Yes 1	No 2	
If **yes**, Impact:	None 3	Some 4	Significant 5		
8) You became unemployed or you were seeking work unsuccessfully for more than one month		Yes 1	No 2 **go to next question**		59
8a) Did this occur in the past 6 months?			Yes 1	No 2	
If **yes**, Impact:	None 3	Some 4	Significant 5		
8b) Did this occur more than 6 months ago?			Yes 1	No 2	
If **yes**, Impact:	None 3	Some 4	Significant 5		
9) You were fired from your job		Yes 1	No 2 **go to next question**		64
9a) Did this occur in the past 6 months?			Yes 1	No 2	
If **yes**, Impact:	None 3	Some 4	Significant 5		
9b) Did this occur more than 6 months ago?			Yes 1	No 2	
If **yes**, Impact:	None 3	Some 4	Significant 5		68

SECTION 7: PSYCHOSOCIAL

Office use

					Office use
10) You had a major financial crisis		Yes 1	No 2 go to next question		69
10a) Did this occur in the past 6 months?			Yes 1	No 2	
If **yes**, Impact:	None 3	Some 4	Significant 5		
10b) Did this occur more than 6 months ago?			Yes 1	No 2	
If **yes**, Impact:	None 3	Some 4	Significant 5		
11) You had problems with the police and a court appearance		Yes 1	No 2 go to next question		74
11a) Did this occur in the past 6 months?			Yes 1	No 2	
If **yes**, Impact:	None 3	Some 4	Significant 5		
11b) Did this occur more than 6 months ago?			Yes 1	No 2	
If **yes**, Impact:	None 3	Some 4	Significant 5		
12) Something you valued was lost or stolen		Yes 1	No 2 go to next question		79
12a) Did this occur in the past 6 months?			Yes 1	No 2	
If **yes**, Impact:	None 3	Some 4	Significant 5		
12b) Did this occur more than 6 months ago?			Yes 1	No 2	
If **yes**, Impact:	None 3	Some 4	Significant 5		83

SECTION 8: ALCOHOL USE			Office use	
	STUDY NUMBER:			4
8A	Do you consume drinks that contain alcohol such as beer, wine, spirits or sorghum beer?	Yes 1 / No 2		5
	If No, go to Section 9			
8B	Have you ever felt that you should cut down on your drinking?	Yes 1 / No 2		
8C	Have people annoyed you by criticizing your drinking?	Yes 1 / No 2		
8D	Have you ever felt bad or guilty about your drinking?	Yes 1 / No 2		
8E	Have you ever had a drink first thing in the morning to steady your nerves or get rid of a hangover?	Yes 1 / No 2		9

SECTION 9: TOBACCO USE				Office use
9A	Do you **currently smoke** any tobacco products, such as cigarettes, cigars, or pipes? **If No, go to question 9F**	Yes 1	No 2	10
9B	Do you currently smoke tobacco products **daily**? **If No, go to question 9F**	Yes 1	No 2	
9C	**How old** were you when you first **started** smoking daily? Years old Don't remember/not sure = 99			
9D	If you do not remember how old you were, do you remember how long ago you started smoking daily?			
	1 WEEKS AGO			
	2 MONTHS AGO			
	3 YEARS AGO			16
9E	On average, how many of the following items do you smoke each day? [NONE = 00]			
	1 nufactured cigarettes?			
	2 Hand-rolled cigarettes?			
	3 Pipes full of tobacco?			
	4 Cigars/Cheroots/Cigarillos?			24
	Go to question 9I			

SECTION 9: TOBACCO USE				**Office use**	
9F	**In the past,** did you ever smoke daily? **If No, go to question 9I**	Yes 1	No 2		25
9G	**How old** were you when you first **stopped** smoking daily? Years old Don't remember/not sure = 99				
9H	If you do not remember how old you were, do you remember how long ago you stopped smoking daily?				
	1 WEEKS AGO				
	2 MONTHS AGO				
	3 YEARS AGO				30
	ASSESSING USE OF SMOKELESS TOBACCO				
9I	Do you **currently use any** smokeless tobacco, such as snuff or chewing tobacco? **If No, go to question 9L**	Yes 1	No 2		
9J	Do you currently use smokeless tobacco **daily**? **If No, go to question 9L**	Yes 1	No 2		
9K	On average, how many times do you use each of the following items per day? [NONE = 00]				
	1 Snuff (bymouth)?				34
	2 Snuff (by nose)?				
	3 Chewing tobacco?				
	Go to Section 10				
9L	**In the past,** did you ever use smokeless tobacco, such as snuff or chewing tobacco daily?	Yes 1	No 2		40

SECTION 10: PHYSICAL ACTIVITY – MODIFIED STEPS/GPAQ	*Office use*
STUDY NUMBER:	4
10 The next questions are about the time you spend doing different types of physical activities. This includes activities you do **at home, at work, travelling from place to place and during your spare time**. You are requested to answer the questions even if you don't consider yourself to be an active person.	
Occupation-Related Physical Activity (paid or unpaid work): When answering the following questions, think back over the **past 12 months** and consider (think of) **a usual week**.	
10A Does your work involve vigorous activities, (like heavy lifting, digging, or heavy construction) for **at least 10 minutes** at a time? Yes 1 / No 2 **If No, go to question 10D**	
10B In **a usual week**, how many days do you do vigorous activities as part of your work? DAYS:	
10C On **a usual day** on which you do vigorous activities, how much time do you spend doing such work? 1 HOURS: 2 MINUTES:	
10D Does your work involve moderate-intensity activities, (like brisk walking or carrying light loads) for **at least 10 minutes** at a time? Yes 1 / No 2 **If No, go to question 10G**	13

SECTION 10: PHYSICAL ACTIVITY – MODIFIED STEPS/GPAQ	*Office use*

10E In **a usual week**, how many days do you do moderate-intensity activities as part of your work?

DAYS: 15

10F On **a usual day** on which you do moderate-intensity activities, how much time do you spend doing such work?

1 HOURS:

2 MINUTES:

Travel-Related Physical Activity: Other than activities that you've already mentioned, I would like to ask you about the way you travel to and from places (to work, to shopping, to market, to church, etc.).

10G Do you walk or use a bicycle (pedal cycle) for **at least 10 minutes** at a time to get to and from places?

Yes 1 | No 2

If No, go to question 10J

10H In **a usual week**, how many days do you walk or cycle for at least 10 minutes to get to and from places?

DAYS:

10I On **a usual day,** how much time do you spend walking or cycling for travel?

1 HOURS:

2 MINUTES:

Non-Work Related and Leisure Time Physical Activity: The next questions ask about activities you do in your leisure or spare time, for recreation or fitness. Do not include the physical activities you do at work or for travel already mentioned.

10J In your leisure or spare time, do you do any vigorous activities (like running or strenuous sports, weightlifting) for **at least 10 minutes** at a time?

Yes 1 | No 2

If No, go to question 10M

10K In **a usual week**, how many days do you do vigorous activities as part of your leisure or spare time?

DAYS:

10L How much time do you spend doing this on **a usual day?**

1 HOURS:

2 MINUTES: 38

10M In your leisure or spare time, do you do any <u>moderate-intensity</u> activities (<u>like</u> brisk walking, cycling or swimming) for **at least 10 minutes** at a time? Yes 1 No 2

If No, go to question 10P

10N In **a usual week**, how many days do you do <u>moderate-intensity</u> activities as part of your leisure or spare time? DAYS: 42

10O How much time do you spend doing this on **a usual day?**

1 HOURS:

2 MINUTES:

Sitting / Resting Activity: Now I would like to ask you about the time spent sitting or resting, not including sleeping, **in the <u>past 7 days</u>**. This may include time sitting at a desk, visiting friends, reading, or sitting down to watch television **during working hours and leisure or spare time**.

10P Over the **past 7 days,** how much time did you spend sitting or reclining (lying) on **a usual day (excluding sleeping)?** 1 HOURS:

2 MINUTES: 52

SECTION 11: DIETARY HISTORY	***Office use***
STUDY NUMBER:	4
24-HOUR RECALL RECORDING SHEET	
INTERVIEWER'S NAME	
DATE OF INTERVIEW: D D M M Y Y Y Y	
Tick the day of the week you are recalling	
Sunday 1 | Monday 2 | Tuesday 3 | Wednesday 4 | Thursday 5 | Friday 6 | Saturday 7	16

Steps for the Interviewer to follow when interviewing each participant

Step 1: You can start the dietary interview as follows: "I want you to think back to when you woke up yesterday morning. What time was it? Now I want you to try to remember what you ate and drank yesterday from the moment you woke up until you went to sleep again last night. Run through the whole day in your mind and try to remember everything you ate and drank."

You must now give the participant time to remember - during this time you must not disturb them.

Now you can carry on and ask the participant to tell you what he/she ate and drank yesterday, starting in the morning after you got up. .

What was the first thing you ate after you woke up……what time was that? And then……what time was that? And then……what time was that?

You don't have to remember the exact time; tell me if it was for breakfast, lunch, supper, between breakfast and lunch, between lunch and supper or after supper.

Step 2: To check whether the participant forgot anything, ask him / her the following questions:

- Did you have any cold drinks or other drinks yesterday?
- Did you have any sweets and or chocolate yesterday
- Did you have any cake yesterday?
- Did you have any biscuits yesterday?
- Did you have any rusks yesterday?
- Did you have any savoury snacks like chips / popcorn / salty biscuits yesterday?
- Did you have any other fruit yesterday?
- Did you have any other vegetables yesterday?
- Did you have cheese yesterday?
- Did you have any other bread or rolls yesterday?

Step 3			
3a What you ate or drank yesterday was it?	The **same** as usual	**More than** usual	**Less than** usual
3b What type of **milk** is usually used in your home?			
3c What type of **butter or margarine** is usually used in your home?			
3d What type of **oil** is usually used in your home?			

Step 4: Now I am going to ask you more about each food item that you ate yesterday. First, I want you to describe the food item and tell me how it was prepared.

Step 5: Now I want to know how much of each food item you ate. (portion sizes)
You will then start using the food photo manual to obtain the relevant information

STEP 1: Time	STEP 1: Foods eaten	STEP 4: Description of the food item	STEP 5: Description of the portion size

STEP 2: Time	**STEP 2:Forgotten foods**		

MEASUREMENTS			Official use:	
STUDY NUMBER:				4
Anthropometer's name: …………………………………………………….				
Weight:		kg		10
Height:		cm		
Waist circumference:		cm		
Hip circumference:		cm		22
Discard 1st blood pressure reading	1st Systolic blood pressure :	mmHg		
1st Diastolic blood pressure :		mmHg		
1st Heart rate :				
2nd Systolic blood pressure :		mmHg		25
2nd Diastolic blood pressure :		mmHg		
2nd Heart rate :				
3rd Systolic blood pressure :		mmHg		34
3rd Diastolic blood pressure :		mmHg		
3rd Heart rate :				40

CRIBSA: BLOOD RESULTS		**Official use:**	
Name:………………………………………………………………..	Study number		4
Oral glucose tolerance test (OGGT) (mmol/ℓ)	Fasting glucose 0 minutes		
75 g oral glucose:	Blood glucose 30 minutes		
	Blood glucose 120 minutes		13
Insulin (MIU/mℓ)	0 minutes		
Insulin (MIU/mℓ)	30 minutes		
Insulin (MIU/mℓ)	120 minutes		22
	Total cholesterol (mmol/ℓ)		
	HDL cholesterol (mmol/ℓ)		28
	Triglycerides (mmol/ℓ)		
	LDL cholesterol (mmol/ℓ)		34